Proctor, C.J., Warren, N.D., Bevan, M.A.J. and Baker-Rogers, J. (1991)[20]

> It seems that the main factor in exposure to environmental tobacco smoke is living with a smoker. Exposure at work, leisure, or travel seems to be minor. Overall, exposure to airborne nicotine was found to be low (mean 2.3 $\mu g\ m^{-3}$, median $<0.1\ mg\ m^{-3}$). (p. 296)

Mennear, J.H. (1993)[21]

> The role, if any, of environmental tobacco smoke (ETS) in the causation and/or exacerbation of cardiovascular disease remains to be proven and defined. . . . The findings show that there is scant clinical or experimental evidence to support a role for carbon monoxide in the causation of ischemic heart disease. Further, the results of field studies of relative air quality in nonsmoking and smoking homes, offices, and public places show that ETS contributes only minor and toxicologically insignificant increments in ambient carbon monoxide concentrations. These increments are variable and easily masked by other common carbon monoxide sources such as internal combustion engines and the burning of cooking and heating fuels. It is concluded that if ETS plays a role in the etiology of cardiovascular disease, it is most likely not mediated through carbon monoxide. (p. 77)

Roe, F.J.C., (1993)[22]

> Although it has not been proven that intermittent low peak levels of COHb do no permanent harm, it seems likely that the healthy body can compensate for them completely. (p. 120)

There is no clear evidence that exposure to CO is associated with an increased incidence of any form of cardiovascular disease. (p. 125)

Rylander, R. (1983)[23]

The unimportance of carbon monoxide has been further confirmed. . . . CO from ETS is not important from a health point of view. (p. 144)

Schievelbein, H. and Richter, F. (1984)[24]

Cardiovascular effects of tobacco smoke have been studied in passive smokers far less extensively than in active smokers. Under real-life conditions, passive smokers inhale approximately 0.02 to 0.01 of the amount of particulate matter taken up by active smokers. Their nicotine concentration in serum is within a range that is barely distinguishable from the background level. The increase in carboxyhemoglobin rarely exceed 1%. In healthy subjects heavily exposed to tobacco smoke, no or only slightly acute effects on the cardiovascular system are found. Whether or not passive smoking is likely to aggravate symptoms in patients with advanced coronary heart disease has not yet been unequivocally established and requires further investigation. From a few studies on occupational groups exposed to carbon monoxide (CO) and from experiments with animals chronically treated with CO or nicotine, the conclusion can be drawn that neither CO nor nicotine is likely to play a role in the development and progression of coronary heart disease in those concentrations normally found in passive smokers. (p. 626)

C. Literature emphasizing that any measurable acute cardiovascular responses to ETS are minor

(1) Healthy subjects

Several aspects of cardiovascular function, such as heart rate and blood pressure, can be readily measured in laboratory studies. Even if short-term changes in such parameters were consistently reported following acute exposure to ETS or CO, their potential clinical significance is highly uncertain. On the other hand, if the literature does not consistently report important acute cardiovascular responses, this further undermines claims of a potential relationship between ETS and heart disease.

A number of studies and reviews have examined potential acute cardiovascular responses to either ETS or CO. A sample of this literature is cited below, indicating a sizeable body of scientific data and opinion questioning a role of ETS or CO in such effects.

Committee on Passive Smoking, Board on Environmental Studies and Toxicology (1986)[25]

The report reviews studies of possible acute effects of ETS on the cardiovascular system in healthy subjects, and concludes that there are no significant changes. The report, although avoiding a firm conclusion, expresses that, for angina patients,

there is a possible cause for concern about exposure to the CO in ETS.

> In summary, for normal young adult males and females, no significant acute effects of ETS exposure on heart rate or blood pressure have been reported, either under resting or aerobic conditions. (p. 260)

Fischer, T., Weber, A. and Grandjean, E. (1978)[26]

Potential subjective, irritant and physiological (e.g., heart rate) effects of ETS were assessed in a laboratory setting. No pulmonary function or heart rate effects were reported. However, the authors nevertheless consider ETS a serious problem in view of irritant effects, potential effects in sensitive persons and levels to which some restaurant and bar employees are exposed.

> Pulmonary function tests should give us an indication of eventual bronchial constriction, encountered frequently in smokers. In fact, our spirometric methods showed no differences before and after exposure to smoke. The change in heart rate during the test was just as insignificant. (uncertified translation, p. 8)

Harke, H.-P. and Bleichert, A. (1972)[27]

> Measurements of electrocardiogram, blood pressure, and puls [sic] frequency showed, that these parameters were not altered by passive smoking. (p. 312)

Hulka, B.S. (1988)[28]

> Studies of acute ETS exposure in healthy children and adults have shown no statistically significant alterations in heart rate or blood pressure either during resting conditions or during exercise. Studies of ETS exposure in persons with pre-existing atherosclerotic heart disease have been inconclusive. Important questions about ETS exposure and the induction of angina, electrocardiographic abnormalities, and cardiac arrhythmias are unanswered. (p. 537)

International Collaborative Group (1984)[29]

This study examined children (approximately 13 years old) from cities in Germany, Cuba, Hungary and the USSR. Two sample groups were compared, one representing the upper 5% and one representing the remainder of the blood pressure distribution curve. One of the characteristics compared between these two groups was parental smoking habits. No significant association was reported between the children's blood pressure and parental smoking.

> The proportion of smokers among the fathers was slightly higher in the lower (55.8%) than in the upper group (54.7%); this difference was not significant. The proportion of smokers among mothers was, however, significantly higher in the lower than in the upper group ($P<0.01$). The proportion of smoking fathers was lowest in Berlin-Köpenick (46.3% vs. 38.6%) and highest in Havana (66.3% vs. 66.7%). The proportion of smoking mothers was lowest in Moscow 3 (0% vs. 1.4%) and highest in Budapest (29.4% vs. 32.0%). The mean blood pressure values of the children

showed no significant association with the smoking habits of either parent. (p. 122)

Pimm, P.E., R.J. Shephard and F. Silverman (1978)[30]

It is concluded that in normal subjects the magnitude of physiological responses to acute exposures is minimal. . . . (p. 201)

Rummel, R.M., Crawford, M. and Bruce, P. (1975)[31]

Heart rate and blood pressure measurements were made on 17 male and 39 female nonsmoking college students at 5, 10, 15, and 20 minutes during exposure to exhaled cigarette smoke in an enclosed room. . . . Measurements were correlated with attitudes toward smoke exposure.

For the entire group of nonsmokers, there were no significant changes in heart rate or diastolic blood pressure during the smoke exposure, but systolic blood pressure was significantly increased at 5 minutes of exposure; at 20 minutes of exposure it had returned to preexposure values.

When results were examined according to nonsmokers attitudes, those who "disliked" being exposed had significantly greater heart rates than those who were "indifferent" at all measurements; blood pressures between the two groups did not differ significantly.

Samet, J.M. (1988)[32]

> While these effects [relating to oxygen transport, heart and blood pressure increases, etc.] of carbon monoxide and nicotine may impair performance, exposures to environmental tobacco smoke are generally at concentrations below which physiological effects would be expected. (p. 12, col. 1)

Shephard, R.J., Collins, R. and Silverman, F. (1979)[33]

The responses of healthy men and women were measured during exercise performance while exposed to ETS vs. a sham exposure. Among several other variables, heart rate was measured. During a preexposure period, those subjects who were to undergo ETS exposure had higher heart rates than the sham subjects. However, actual exposure was associated with a smaller heart rate increase compared to the sham condition.

> The heart rate was higher before the experimental than before the sham exposures. . . . However, while actually exposed to the cigarette smoke both the increment of heart rate and the absolute heart rate were less than in the corresponding sham exposure. (p. 285)

The authors suggested that these heart rate changes might be related to a subjective anxiety or hyperventilation reaction, rather than an actual physiological response to ETS.

Turner, J.A.McM. and McNicol, M.W. (1993)[34]

In a partially blind randomized control study, nine healthy adult men performed progressive sub-maximal exercise tests, either after chewing nicotine chewing gun (4 mg) or breathing carbon monoxide (320 p.p.m.). Neither agent significantly affected exercise performance, despite a mean rise in blood nicotine to 17.0 ng ml^{-1} during nicotine administration and a COHb rise to 6.9% whilst breathing carbon monoxide. (p. 427)

Weber-Tschopp, A., Fischer, T. and Grandjean, E. (1976)[35]

Thirty three subjects were exposed in a climatic chamber to cigarette smoke (side stream) produced by a smoking machine. . . . $\%FEV_1/VC$, MMF and heart rate were not significantly affected during exposure. (p. 277)

Weber, A., Fischer, T. and Grandjean, E. (1979)[36]

In a first study subjects were exposed for 1 hr to constant cigarette smoke concentrations corresponding to 5 or 10 ppm CO. Annoyance, subjective eye irritations, and eye blink rate increase in both conditions during the first 30 min of exposure. Respiratory frequency and heart rate variability are not altered. (p. 205)

Winneke, G., Plischke, K., Roscovanu, A. and Schlipkoeter, H.-W. (1984)[37]

Neither blood pressure-values, nor heart-rates or fingerpulse-volume were influenced by exposure to tobacco-smoke. This correspond to the fact that nicotine-intake from passive smoking is negligible. Depth and rate of breathing were not altered either. (p. 353)

> As for carbon monoxide (CO) there was pronounced uptake in terms of COHb for the high exposure-condition only. In absolute terms, however, the measured values correspond to those found in non-smoking urban populations, and are well below levels considered critical for persons with cardio-vascular impairment. (p. 354)

Winneke, G., Neuf, M., Roscovanu, A. and Schlipköter, H.-W. (1990)[38]

Physiological measurements were obtained from nonsmokers, while they were in an exposure chamber in which another individual was smoking. No significant effects were reported for heart rate or blood pressure. The authors reported that "cardiorespiratory variables were not affected by ETS exposure." (p. 173)

> In taking CO as the basis for comparison our cardiovascular findings are consistent with those of others, who, at even higher levels of ETS-exposure did not observe exposure-related increase of either heart-rate or blood-pressure. Nicotine-intake at such ETS-levels is likely to be too low for cardiovascular changes to be expected. (p. 181)

(2) Angina and other heart disease patients

Claims are sometimes made that ETS, or specifically CO in ETS, aggravates cardiovascular disease in people with pre-existing illness. Such claims typically stem from a limited and largely discredited report by Wilbert Aronow that angina patients

experienced chest pain sooner when exercising in the presence of ETS.[39] However, a closer examination of the literature reveals a body of data that raises questions about whether ETS, or CO at levels reported to be in ETS, has significant adverse cardiovascular effects.

Hinderliter, A.L., Adams, K.F., Price, C.J., Herbst, M.C., Koch, G. and Sheps, D.S. (1989)[40]

> In conclusion, low-level CO exposure is not arrhythmogenic in patients with coronary heart disease and no ventricular ectopy at baseline. (p. 89)

McNicol, M.W. and Turner, McM. (1983)[41]

> There was no change in the mean oxygen uptake at the onset of angina with any intervention. . . . These results are in contrast with previous reports of the effects of smoking and carbon monoxide on exercise performance in angina pectoris. However in all of these other studies a subjective rather than objective end point was used. We suggest that an objective assessment is essential and that oxygen uptake at the onset of angina is useful and relatively easy to measure.

Shephard, R.J., Collins, R. and Silverman, F. (1979)[42]

This study was an attempt to determine if asthmatic subjects have an increased sensitivity to ETS exposure. Respiratory symptoms, pulmonary function and heart rates were measured. Although some slight effects (e.g., a small heart rate increase) were reported, these were considered to be of "doubtful

biological importance" and most likely due to an emotional, rather than directly physiological, response to the ETS exposure.

> There was also some evidence of arousal and/or emotional excitement, including a slight tachycardia (at 80-min exposure, $P<0.05$) and a slight increase of forced vital capacity ($P<0.05$ at 90-min exposure). However, dynamic lung volumes . . . were unaltered. . . . Our data thus do not suggest that asthmatic subjects have an unusual sensitivity to cigarette smoke. (p. 392)

> The physiological changes observed in normal subjects during smoke exposure, although occasionally reaching conventional levels of statistical significance, were of doubtful biological importance (Pimm et al., 1978; Shephard et al., 1979a). Findings included some increase of heart rate and respiratory minute volume, probably of emotional origin, a tendency of increase in functional residual capacity and residual volume in some experiments, and small decreases of dynamic lung volumes. The asthmatic subjects also showed emotional reactions to the cigarette smoke, including the tachycardia, and possibly the preexposure increase of FRC and TLC. (pp. 399, 401)

> We would thus conclude that the specific sensitivity of asthmatic subjects is not a major consideration when determining air quality criteria for rooms contaminated by cigarette smoke. (p. 402)

Sheps, D.S., Adams, K.F., Bromberg, P.A., Goldstein, G.M., O'Neil, J.J., Horstman, D. and Koch, G. (1987)[43]

> In conclusion, there is no clinically significant effect of 3.8% COHb (representing a 2.2% increase from resting values) on the cardiovascular system in this study. (p. 108)

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SECTION VII

MATERIAL IMPAIRMENT: IRRITATION

IRRITATION

THE PROPOSED RULE DOES NOT CORRELATE COMPLAINTS OF IRRITATION WITH ACTUAL WORKPLACE EXPOSURES TO ETS, AND FAILS TO DEMONSTRATE THAT CURRENT ETS EXPOSURE LEVELS IN THE WORKPLACE POSE A SIGNIFICANT RISK OF MATERIAL IMPAIRMENT FROM "IRRITATION"

OSHA's Proposed Rule states that "mucus membrane irritation" is part of "a wide spectrum of health effects . . . associated with exposure to ETS." (59 FR 15973) In support of its claim that ETS is an "irritant," OSHA cites a single summary review of the literature (1986), two studies and six personal testimonials submitted to the RFI docket. (59 FR 15975) The Proposed Rule also lists several substances that have been defined as "irritants" by NIOSH and are reportedly found in tobacco smoke, but it concedes that specific constituents in ETS have not been directly related to irritation effects. (59 FR 15987-8, 15975)

The two studies on ETS and irritation cited in the Proposed Rule do not establish a significant risk of material impairment at current workplace exposure levels. The data in one of the studies cited in the Proposed Rule were collected in Switzerland in the late 1970s, and the high levels of respirable particulate matter reported therein are not relevant to current ETS exposure levels in the U.S. (Ex. 4-317) Moreover, the Proposed Rule does not address the authors' qualifications of the data in the study, i.e.,

> . . . there is a very poor correlation between the mean pollutant concentrations in each room and the corresponding results of the interview. This means that the measured pollutant concentration in an individual work room does not necessarily allow drawing conclusions on the extent of disturbance and irritations . . . .;
>
> Individual psychological factors (relationship with co-workers, work satisfaction, attitude toward smoking) considerably influence individual evaluation of disturbances and irritations . . . .;
>
> Thus, there is a considerable variance on the side of measurements of chemical and physical properties as well as on the side of subjective perception and feelings.

A second study cited in the Proposed Rule was conducted in Japan in the mid-1980s. (Ex. 4-18) Subjects were exposed to extremely high levels of tobacco smoke in an experimental chamber. The peak level of exposure to respirable particulate matter was nearly 100 times greater than levels encountered in workplace situations where smoking is permitted. The relevance of this experimental study to OSHA's claim about irritation is unclear; Benzene explicitly prohibits OSHA from basing its finding of "significant risk" at "lower levels of exposure . . . using evidence of health impairments at significantly higher levels of exposure." (AFL-CIO v. OSHA, 965 F.2d 962, at *978)

"Irritation" Defined

In the introduction to its standard governing workplace exposure to air pollutants, OSHA summarizes its authority as follows:

> Some impairments are so slight a discomfort that they are not material and do not provide a basis for regulation. A complaint of minor discomfort, in and of itself, is not material impairment. However, the OSHA Act is designed to be protective of workers and is to protect against impairment with less impact than severe impairment. (54 FR 2332, 1989)

OSHA also states:

> . . . [C]omplaints of minor irritation would not in and of itself constitute material impairment. In addition, OSHA would weigh irritation with physical manifestations more heavily than irritation with purely subjective responses. This does not mean that purely subjective responses would not constitute material impairment. That judgment would depend on the magnitude of the irritation. (54 FR 2362, 1989)

If OSHA is to support its claim that irritation from exposure to ETS is a material impairment, it must demonstrate that exposures bring about something more than subjective responses of minor irritation or annoyance, i.e., it must demonstrate the "magnitude" of the purported irritation effect. OSHA must demonstrate the "extent of the risk posed by individual substances," i.e., that existing exposure levels in the workplace

pose a significant risk of material impairment (due to irritation). It also must provide an "assessment of the level at which significant risk of harm is eliminated or substantially reduced." (AFL-CIO v. OSHA, 965 F.2d 962, 1992; *974, *979, *975)

OSHA will find little, if any, support for its position in the scientific literature. For instance, the major databases on sick-building syndrome indicate that tobacco smoke is rarely the underlying cause of complaints about irritation and annoyance related to indoor air quality. (Exs. 3-1053, 3-1073, 3-1074). ETS has been identified as a source of claimed discomfort in only two to five percent of a combined total of 1,000 sick-building investigations.[1-3]

Other published investigations of workplace venues report similar results. For example, in 1992, Canadian researchers reported results of a building investigation in which temperature, humidity, dust, nicotine, formaldehyde, volatile organic compounds, and CO_2 were measured.[4] Occupants of the building complained of poor indoor air quality, and a questionnaire evaluation revealed a substantial number of complaints about unsatisfactory thermal conditions, dry air, drowsiness and eye irritation. However, all measured parameters, including temperature and humidity, were within accepted comfort/exposure guidelines. The authors were unable to correlate any single measured environmental parameter

with complaints or symptoms. They concluded that their investigation "showed that complaints reported by occupants were associated with perceived rather than measured levels of indoor environmental parameters" [emphasis added].

A 1989 report by Hedge, et al. noted that while self-reported exposures to ETS were associated with increases in perceived symptoms of irritation, no significant correlations between actual levels of ETS constituents in the indoor air and symptom prevalence could be established. Moreover, the researchers reported that "passive smoking" was not associated with self-reports of mucus membrane irritation (MMI).[5]

Pimm and co-workers examined the effects of ETS exposures "at levels typically encountered in public buildings" in the late 1970s.[6] Physiological parameters such as lung flow rates, heart rates and blood carboxyhemoglobin levels were measured. The researchers reported that "the magnitude of the changes was small and of questionable biological significance." The authors also observed that "the subjects were also inevitably aware of the presence of the smoke, and the reported reactions may have been influenced by underlying beliefs and attitudes." Subjective complaints of irritation were categorized as "symptomatic" rather than physiological.

The Proposed Rule rejects the possibility that complaints about indoor air quality, and specifically complaints about ETS, may be mediated by a psychological component. The Proposed Rule suggests only that a "psychological overlay" may be common to complaints related to poor indoor air quality. (59 FR 15970) However, research indicates that the mere visibility or presence of ETS may provoke symptoms and complaints.[7,8] Winneke and colleagues found that aversion to ETS increased the degree of annoyance and irritation reported under experimental exposure conditions.[7] Researchers from the Illinois Institute of Technology in 1992 reported that simple visual contact "with a source of tobacco odors" increased both perceived odor intensity and the number of subjects perceiving tobacco odor.[8]

Other recently published studies have demonstrated the importance of variables such as job stress, including work control, support, possibilities for personal growth and job satisfaction, in the perception of symptoms due to sick-building syndrome.[9,10] The studies suggest that such factors may either be directly related to symptoms as stress reactions, or they may play a role in rendering the individual more prone to react to environmental influences, including perceived exposures to ETS.[9,10]

ETS is easily identified and is often blamed as the cause of complaints of annoyance and irritation, especially among

individuals who dislike tobacco smoke. However, a workplace that is adequately ventilated with outdoor air effectively addresses and prevents complaints of irritation and annoyance, regardless of the suggested source.[1-3] ASHRAE ventilation Standard 62-1989 was developed in order to minimize potential complaints of annoyance and irritation in indoor environments. The ventilation rates in the Standard specifically address ETS. The Standard's design criteria currently apply to buildings that have been constructed, renovated or remodeled in the U.S. after 1990. Indeed, the Standard is incorporated by reference into OSHA's Proposed Rule on IAQ. (59 FR 16036)

OSHA's Proposed Rule calls for the complete elimination of ETS exposure in the workplace. This proposal, of course, will not eliminate exposure to irritants in the workplace, since precisely the same irritants imputed to tobacco smoke by OSHA are emitted from other sources. (59 FR 15987-8, 15984) Specific irritants reported in tobacco smoke and generated as well from other indoor sources include formaldehyde and other volatile organic compounds such as acetaldehyde, acrolein, etc. 59 FR 15987-8)

The levels of "irritants" found in the indoor air of the nonindustrial workplace typically overwhelm any possible contributions from tobacco smoke. For example, Godish, in his

review of formaldehyde exposures from tobacco smoke, writes that even under "extreme circumstances, the effect of cigarette smoking on formaldehyde levels in indoor spaces would be negligible.[11] Guerin, et al., similarly conclude that "as such, it has been difficult for many studies to demonstrate consistently elevated levels of formaldehyde due to ETS."[12] (Ex. 8-129, also submitted to OSHA as Ex. 3-499) They also conclude: "Studies of offices, restaurants, train compartments, and public buildings suggest that ETS contributes to the indoor air burden of VOCs but that other sources predominate. Major sources include building materials, furnishings, cleaning products, office machines, gasoline, and combustion sources associated with cooling, heating, and transportation."[12] Clearly, these results indicate that contributions from ETS to ambient levels of "irritants" are minimal and often indistinguishable from background levels. Any potential risk from irritation associated with ETS therefore would be addressed with the implementation of OSHA's own Proposed Rule on indoor air quality (a ventilation-based indoor air quality standard).

The scientific record is devoid of any data suggesting that ETS is an irritant under current workplace exposure levels and/or under conditions of adequate ventilation (e.g., outdoor air rates specified in ASHRAE 62-1989). OSHA's proposal to reduce alleged irritation from ETS through the complete elimination of

smoking in the workplace is equivalent to the "regulation of insignificant risks." Benzene clearly states that reduction of significant risk is not tantamount to elimination of all "risk," but only the level or range that may be deemed "significant." (IUD v. API, 448 U.S. 649-651) OSHA has failed to determine any level of that purported "risk."

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SECTION VIII

MATERIAL IMPAIRMENT: PULMONARY EFFECTS

PULMONARY EFFECTS

OSHA DOES NOT EXERCISE ANY CONSISTENCY OR PURPOSE IN ITS CITING OF THE PUBLISHED LITERATURE; STUDIES ARE CITED FOR IRRELEVANT OR MINOR POINTS, WHILE PERTINENT DATA FROM THE STUDIES ARE NOT REPORTED OR DISCUSSED; OSHA DOES NOT PRESENT ANY CONCRETE ARGUMENTS TO SUPPORT THEIR CONTENTIONS

This is a consistent problem throughout this section of OSHA's Proposed Rule on IAQ/ETS. It appears as though OSHA chooses irrelevant points from studies so as to be able to reference them, while the pertinent data from the studies are ignored in the discussion. No critical analysis is presented, and OSHA makes no concrete arguments to support its conclusion that there is an association between exposure to ETS in the workplace and pulmonary effects in nonsmokers.

OSHA presents data which are irrelevant to a discussion of potential pulmonary effects in nonsmokers exposed to environmental tobacco smoke in the workplace

OSHA's task is to show that there is a significant risk of material health impairment in nonsmokers exposed to environmental tobacco smoke in the workplace. OSHA, however, bases its "review" of pulmonary effects on one experimental study (Ex. 8-18) in humans as well as on several epidemiological studies (Exs. 8-37, 8-62, 8-148, 8-173, 8-176, 8-178, 8-180, 8-209, 8-210, 8-278, 8-

295, and 8-321), most of which examined reported exposures to ETS in the home environment. OSHA also discusses several animal inhalation studies. (Exs. 8-322, 8-204, 8-290, 8-104, 8-45, and 8-114) OSHA concludes the following:

> The weight of the evidence shows that exposure to ETS results in decreases in pulmonary function indices and increases in respiratory symptoms in otherwise healthy men and women who are exposed to ETS for periods of 10 or more years. [emphasis added] (59 FR 15977)

Because there are studies available that have examined potential effects of exposure to ETS on the respiratory health of nonsmoking adults in the workplace, those are the studies which should be reviewed and analyzed in this section. OSHA presents no critical discussion of the data available from these studies and makes no scientifically supportable claims regarding causation. Recent reviewers of the pulmonary effects literature (who are affiliated with the International Agency for Research on Cancer) concluded that "on the basis of the available data, no definite conclusion (excluding the acute irritating effect of ETS on respiratory mucous membranes) can be drawn."[1] The reviewers' conclusions were not discussed in the Proposed Rule.

OSHA relies on an experimental exposure chamber study which did not examine or make any conclusions regarding respiratory endpoints in nonsmokers exposed to ETS

OSHA cites a 1983 study by Asano, et al. (Ex. 8-18) This study was an experimental exposure chamber study of only 20 subjects, including 5 male smokers, 5 male nonsmokers and 10 female nonsmokers. It is unclear why OSHA included this study in the pulmonary effects section, since the study reports on non-pulmonary endpoints such as systolic blood pressure and heart rate. This study is therefore irrelevant to a discussion of potential pulmonary effects in nonsmokers exposed to ETS in the workplace.

OSHA RELIES ON SURVEY REPORTS OF NONSMOKERS WHO LIVE WITH ONE OR MORE SMOKER(S); THESE STUDIES SUFFER FROM IMPORTANT FLAWS, AND DO NOT CONSTITUTE THE "BEST AVAILABLE EVIDENCE" REGARDING POTENTIAL PULMONARY EFFECTS IN NONSMOKERS EXPOSED TO ETS IN THE WORKPLACE

The epidemiological literature upon which OSHA bases its conclusion suffers from a couple of very important problems. First, very few of the studies report on workplace exposures to environmental tobacco smoke, the very issue OSHA is proposing to regulate. Second, the studies contain no actual exposure data. In the majority of studies, the estimation of exposure originates from questionnaire responses of subjects and/or their spouses and co-habitants. Even though the studies suffer from these problems, the magnitude of the reported decrements in pulmonary function in

healthy nonsmokers reportedly exposed to environmental tobacco smoke, as OSHA itself concedes, is very small and of questionable biological or clinical significance.

Among the epidemiologic studies OSHA chose to rely on are several studies of reported home exposures to ETS. The 1985 study by Brunekreef, et al. estimated exposure of nonsmoking women in the Netherlands by asking the subjects to quantify the number of cigarettes smoked daily inside the home by household members. While this study, on cross-sectional analysis, reported that several pulmonary function parameters appeared to be significantly associated with exposure to ETS in the home while others did not, the authors also reported that "there was no association between [ETS] exposure and pulmonary function decline." (Ex. 8-37)

Another study referenced by OSHA, a 1981 paper by Comstock et al., examined reported home exposures of 1,724 adults to environmental tobacco smoke. (Ex. 8-62) The authors of this study, epidemiologists at the Johns Hopkins University School of Medicine, reported:

> The presence of a smoker in the household other than the subject <u>was not associated with the frequency of respiratory symptoms, and only suggestively associated with evidence of impaired ventilatory function</u>. [emphasis added]

A 1989 study by Hole, et al., is also cited by OSHA. (Ex. 8-148) In this paper, "passive smoking" was defined as living at the same address as a smoker. Additional attempts to quantify exposure involved questioning subjects regarding the number of cigarettes smoked daily by their co-habitee. While this study reported associations between living with a smoker (as a surrogate of ETS exposure) and several respiratory health endpoints in nonsmokers, the 95% confidence interval fell below unity for all respiratory endpoints (i.e., the associations were <u>not</u> statistically significant).

In the Letter to the Editor written by Kalandidi in 1987, which informally reports on a study the author was conducting in Greece that suggested exposure to environmental tobacco smoke "may contribute to the cause of COLD [chronic obstructive lung disease]," the author writes that the studies on exposure to ETS and adult respiratory health contain data that are "equivocal." (Ex. 8-173) In 1990, Kalandidi reported these data in a published paper.[2] While the data were suggestive of a possible association, it is questionable whether the authors adequately considered other potential exposures which have been associated with the development of COLD such as the extremely high levels of outdoor air pollution found in Greece.

OSHA cites two studies by Kauffman, et al. The first study, published in 1983, reports on home exposures to ETS of nonsmoking women married to smokers. (Ex. 8-176) The second study, published in 1989, reports on similar data. (Ex. 8-178) While the authors reported an association between decreased lung function and "passive smoking" in French women, the authors reported that "no significant association between passive smoking and level of lung function" was apparent in American women. Dr. Michael Lebowitz (a member of the EPA's Science Advisory Board Committee which reviewed the EPA's Risk Assessment on ETS) has questioned the conclusions of the Kauffman study (Ex. 8-176), which, as mentioned above, reported significant differences in lung function between exposed and nonexposed nonsmokers in one part of the study population but no significant differences in the population as a whole. Dr. Lebowitz noted that since the "healthiest" part of the study population lived in the most polluted areas, the study may have been flawed due to biased population selection and testing for other confounding factors.[3]

Since OSHA decided to rely on epidemiologic studies of reported ETS exposures in the home, studies of reported home exposures not cited by OSHA in its proposed rulemaking warrant mention.[4-6] In 1983, for example, Jones, et al., reported that in a case-control study of several hundred nonsmoking women from a U.S. study population, there was no significant association between

decreases in lung function and exposure to ETS in the home.[4] Similarly, Lebowitz, et al., coordinators of an epidemiologic study of obstructive lung disease in Arizona, reported no association between reported ETS exposures in the home and respiratory health in their adult study population.[5] In a study of 376 families, Yale University scientists Schilling, et al., also reported no evidence that environmental tobacco smoke affects either lung function or respiratory symptoms in adults.[6]

Similarly, OSHA failed to discuss various government reviews of the ETS and adult respiratory health literature which have generally concluded that there is a negligible association, if any, between exposure to ETS and respiratory health effects in adult nonsmokers. For example, the 1986 Surgeon General's report on ETS concluded:

> Healthy adults exposed to environmental tobacco smoke may have small changes on pulmonary function testing, but are unlikely to experience clinically significant deficits in pulmonary function as a result of exposure to environmental tobacco smoke alone.

Other researchers who have analyzed these reported data contend that the results are "mixed and inconclusive." For example, researchers in France, Laurent, et al., have commented that purported long-term health effects from exposure to ETS are difficult to demonstrate in healthy adults and that the results of

the epidemiological studies are "sometimes conflicting and often open to question."[7] One investigator who is critical of ETS conceded that "the effect of passive smoking on respiratory infections in adults has not been well characterized and reports of its effects on chronic respiratory disease in adults have been inconsistent."[8]

In his summary of the studies on respiratory symptoms and diseases in adults published up to early 1990, American investigator Dr. Philip Witorsch noted that while "4 of 8 [reported] an increased frequency, 4 of 8 [reported] no increased frequency." He stated that, in addition to "all of the problems" with the studies, "these results are too variable to permit any conclusion of association."[9] As his summary demonstrates, the conclusion of the participants at the 1983 U.S. National Institutes of Health workshop on ETS exposure, that the possible effect from ETS "varies from negligible to quite small," is still pertinent.[10]

Thus, OSHA's Proposed Rule has not demonstrated a significant risk of material impairment to health associated with possible pulmonary effects in nonsmokers exposed to ETS in the workplace. Both the literature cited by OSHA and additional material omitted from the Proposed Rule report data which are equivocal and which appear to lack clinical significance.

THE EPIDEMIOLOGIC STUDIES WHICH EXAMINED REPORTED WORKPLACE EXPOSURES TO ETS IN NONSMOKING ADULTS HAVE REPORTED INCONSISTENT AND INCONCLUSIVE DATA; THE STUDY UPON WHICH OSHA PLACES THE MOST WEIGHT HAS RECEIVED SUBSTANTIAL CRITICISM IN THE SCIENTIFIC LITERATURE FOR QUESTIONABLE DATA ACQUISITION AND ANALYSIS; OSHA FAILS TO ADDRESS ANY OF THESE CRITICISMS

OSHA reports on four studies which contain discussions of workplace exposures to environmental tobacco smoke. Probably the most familiar study mentioned in this section is the 1980 White and Froeb study. (Ex. 8-321) While this study is often cited by individuals in support of an association between exposure to environmental tobacco smoke in the workplace and small airways dysfunction in healthy nonsmokers, the study has received substantial criticism in the published literature. OSHA does not address any of these criticisms in its Proposed Rulemaking.

In regard to the White and Froeb study, a physician at a U.S. medical school questioned their use of carbon monoxide as an index of smoke exposure, contending that they "do not have reliable estimates of the smoke exposure in the environment of their nonsmokers" because it is not unique to tobacco smoke.[11] A British reviewer shared the physician's view that White & Froeb's findings "relate to an index which is contentious and certainly not an accepted reliable indicator of an increased health risk."[12] White and Froeb themselves noted that the average values of the pulmonary

tests of nonsmokers exposed to tobacco smoke "were not notably different" from the values suggested as normal by a specialist in this area.[13]

Perhaps the most telling criticisms of the study were voiced by Dr. Michael Lebowitz of the University of Arizona at an annual joint meeting of the American Lung Association/American Thoracic Society and in a subsequent letter published in the U.S. Congressional Record.[14-15] During a forum at the ALA meeting, Dr. Lebowitz stated that he had concluded, from his own extensive review of the study and from an interview with White, that the study was "improperly designed" from an epidemiological point of view. He noted that there were problems "inherent" in the study, including the selection of the study group and the measurement of smoke in the workplace. Dr. Lebowitz also expressed concern that the statistical significance of the data appeared to depend on the unexplained omission of data for 3,000 people who were originally included in the study. Based upon these considerations, Dr. Lebowitz urged that the study not be used to support the claim that exposure to ETS in the workplace affects the lung function of healthy nonsmoking adults.

Dr. Lebowitz again took issue with the White and Froeb study in a 1984 paper he presented at the Vienna Symposium on Passive Smoking.[3] He contended:

> Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.

Fielding and Phenow, whose conclusion that the "changes" reported by White and Froeb were "equivalent to those found in light smokers, who smoke from 1 to 10 cigarettes per day" is reported by OSHA, also made several highly relevant statements that are not mentioned by OSHA. (Ex. 8-102) Fielding and Phenow suggest that the data from the studies on ETS exposure and adults "have been conflicting" and that "taken together, the limited data on the effect of [environmental tobacco] smoke on adult lung function are inconsistent and inconclusive."

In addition to these concerns, the data from the White and Froeb study appear to be inconsistent with other data on lung disease and lung function in nonsmokers. For example, a 1984 study of 1,351 German office workers by Kentner et al., reportedly found no effect of ETS on pulmonary function among exposed nonsmokers. (Ex. 8-180) In a 1988 update of the study, the investigators noted that "there is no evidence that average everyday passive smoke exposure in the office or home leads to an essential reduction of lung function in healthy adults." The key investigator reiterated these conclusions in 1989 and 1990 publications. The 1984 study is

cited by OSHA in its proposed rule, but no discussion of the study or its data is included.

Two other studies cited by OSHA which report data on workplace exposures of nonsmoking adults to ETS suffer from serious problems regarding exposure assessment. The Masi (1988) study reported on 293 subjects, aged 15 to 35 years old. (Ex. 8-209) The study, however, contains such crude "exposure" data that it is disturbing that OSHA deems it reliable. Workplace "exposure" to ETS was assessed in the following manner:

> Bank employees were asked to [subjectively] assess smoke conditions in the work area as light, moderate, heavy or unable to quantify. . . .[these categories] were arbitrarily converted to number of cigarettes. [emphasis added]

Similarly, the authors of the Masjedi (1990) study conceded that most of their subjects "were unable to quantify the amount of [their] exposure to passive smoke. . ." (Ex. 8-210)

THE EPIDEMIOLOGIC AND EXPERIMENTAL DATA AVAILABLE DO NOT SUPPORT OSHA'S CONTENTION THAT COMPROMISED INDIVIDUALS IN THE WORKPLACE HAVE A SIGNIFICANT RISK OF MATERIAL HEALTH IMPAIRMENT BECAUSE OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

OSHA claims that although the pulmonary function decrements reported to be associated with exposure to ETS in

healthy nonsmokers are small and of questionable clinical significance, these changes may become significant in persons with already impaired pulmonary function (e.g., asthmatics). In its attempt to substantiate this claim, OSHA references two experimental studies on adult asthmatics exposed to ETS for one hour in an exposure chamber. The first study, Knight and Breslin, 1985, reported on only six subjects, four of whom had previously given a positive history of ETS "attacks." (Ex. 8-182) The authors (and OSHA) did not discuss the possibility that psychological factors may have played a role in the subjects' reported "reactions" to the ETS exposure.

The second study, by Dahms et al., 1981, reported on ten patients with asthma (five of whom were included because they reported specific complaints when exposed to cigarette smoke) and ten normal patients. (Ex. 8-76) While the authors reported an association between exposure to ETS and lung function responses in the asthmatic subjects, they reported that "not all subjects showed the same pattern of pulmonary responses to the smoke exposure." The authors also reported that "we were not able to exclude the possibility that these changes in pulmonary function were emotionally related to cigarette smoke" [emphasis added]. Similarly, the 1986 Surgeon General's report recognized that "in this study, subjects were not blinded as to the exposure and were selected because of complaints about smoke sensitivity."

OSHA also references a 1991 review article by Stanton Glantz and Richard Daynard (Tobacco Liability Project) which makes several claims regarding exposure to environmental tobacco smoke, none of which is supported by a thorough and critical review of the scientific literature available. (Ex. 3-438D)

OSHA claims that asthmatics are believed to be especially sensitive to various environmental influences, including ETS, but a thorough review of the scientific data on ETS does not convincingly support this contention. There are currently ten major studies on ETS exposures among adult asthmatics. Seven of the studies are clinical in nature, where exposures to ETS were controlled under laboratory conditions. The other three studies are population surveys. All of the studies evaluate reports of respiratory symptoms such as coughing, wheezing, and irritation ("subjective complaints") and changes in lung function (an "objective" test for lung impairment).

Only two of the seven clinical studies report objective decreases in lung function among the majority of asthmatics exposed to environmental tobacco smoke. However, the patients in both positive studies were exposed to excessively high and unrealistic levels of ETS [15-32 ppm carbon monoxide], and, as mentioned above, the authors of one of the studies conceded that the observed effects may have been due to psychological factors. In the

remaining five clinical studies, a number of patients reportedly complained of subjective symptoms upon exposure to ETS, but objective results (i.e., evidence of airways obstruction or significant changes in lung flow rates) were not observed in the vast majority of patients.[16-20]

Data from the three population surveys on adult asthmatics are generally consistent with data reported in the majority of clinical studies.[21-23] A report of a large-scale population survey in the U.S. suggested that ETS exposures in the home did not affect either symptoms or pulmonary function in adult asthmatics. Another group of U.S. researchers recently reported that exposure to ETS did not impair lung function in the 263 asthmatic adult subjects studied. The third study reported mixed results for the development of asthma-related symptoms in individuals reporting exposures to ETS during childhood and adulthood.

OSHA FAILS TO DISCUSS THE PROBLEMS IN GENERALIZING DATA FROM "EXPOSURE CHAMBER" STUDIES TO THE "REAL-WORLD" SITUATION

As mentioned above, the conditions under which asthmatic patients are exposed to ETS in these studies are of questionable relevance to "real-life" exposures. The Surgeon General, in the

1986 report on ETS, conceded that exposure chamber studies may not be indicative of the "real world" situation:

> Acute exposure in a chamber may not adequately represent exposure in the general environment. Biases in observation and the in [sic] selection of subjects and the subjects' own expectations may account for the widely divergent results. Studies of large numbers of individuals with measurement of the relevant physiologic and exposure parameters will be necessary to adequately address the effects of environmental tobacco smoke exposure on asthmatics.

THE MAJORITY OF ANIMAL STUDIES REFERENCED BY OSHA ARE STUDIES OF MAINSTREAM TOBACCO SMOKE AND ARE NOT RELEVANT TO A DISCUSSION OF POTENTIAL PULMONARY EFFECTS IN NONSMOKING ADULTS EXPOSED TO ENVIRONMENTAL TOBACCO SMOKE IN THE WORKPLACE

Although OSHA refers to the studies in this section as experimental "ETS" studies, the majority of the studies are mainstream smoke studies which have little or no relevance to ETS issues. ETS is not the same as either mainstream or sidestream smoke. For a complete discussion of this issue, please refer to the section on exposure issues in this submission.

OSHA mentions isolated conclusions from several experimental animal studies but makes no attempt to explain how these data relate to real-life ETS exposures of nonsmoking adults in the workplace. OSHA also does not make any attempt to explain

the physiological or clinical significance, if any, of the reported data. Nor does OSHA address the apparent inconsistencies in the reported data. For example, "effects" may be reported in one species but not in another.

OSHA makes no attempt to explain the relevance, if any, of these high-dose experiments in animals to real-life workplace exposures in humans, even when some of the studies from which they report data acknowledge that these exposure levels are extreme when compared to the "real world" of "smoke-filled" restaurants or offices. OSHA's responsibility in this proposed rulemaking is to show that workplace exposures to ETS are likely to result in material health impairment in non-smoking adults. They have failed to substantiate this claim.

OSHA FAILS TO REFERENCE MUCH OF THE RELEVANT LITERATURE SUBMITTED IN RESPONSE TO THE OSHA RFI IN 1992

OSHA presents no critical discussion of potential confounding factors, merely stating that the studies "varied" in their consideration of such factors and that "several studies" have examined isolated variables; OSHA makes no statement regarding the adequacy of control for such factors in these studies

In the Proposed Rule, OSHA presents selected data on the possible association between exposure to ETS and decreased pulmonary function in adult nonsmokers. Philip Morris submitted much of the literature which OSHA has omitted from its Proposed Rule to the docket for the RFI in 1992. (Ex. 3-1074) For a discussion of this literature, please see the response to question 2(a)iii in Ex. 3-1074. A discussion of confounding factors, which OSHA does not provide in its Proposed Rule, is also presented in that section of Ex. 3-1074.

OSHA, while conceding that the studies on ETS exposure and pulmonary effects in nonsmoking adults "vary by numerous factors," makes no apparent attempt to explain why these variations are given no importance in their "analysis" of the literature

OSHA does not elaborate on its statement that the studies on ETS exposure and respiratory effects in nonsmoking adults "vary by numerous factors, such as the population studied, the measures

used to estimate exposure to ETS, and the physiologic and health outcomes examined." While other reviewers of this body of literature have concluded that it is difficult, if not impossible, to make any definitive conclusions from these data, OSHA attempts, but fails, to make a case for causation.

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SECTION IX

MATERIAL IMPAIRMENT: REPRODUCTIVE EFFECTS

REPRODUCTIVE EFFECTS

OSHA'S PROPOSED RULE LACKS CONSISTENCY IN ITS DEFINITION OF "ETS EXPOSURE DURING PREGNANCY"

In some instances, OSHA appears to be discussing the potential effects on the fetus that may be associated with active smoking of the mother during pregnancy. At other times, the authors of the Proposed Rule are discussing ETS exposure of the mother during pregnancy. Active smoking of the mother during pregnancy is not synonymous with ETS exposure. OSHA's task is to show that there is a significant risk of material health impairment in fetuses whose mothers are exposed to ETS at work during their pregnancies. The studies referenced by OSHA which examine active smoking during pregnancy are irrelevant to this discussion.

The majority of the studies OSHA references in this section are irrelevant because they deal with active smoking of the mother during pregnancy, not ETS exposure of the mother during pregnancy; ETS and mainstream tobacco smoke are not equivalent

A detailed discussion of the differences between ETS, sidestream and mainstream tobacco smoke is provided in exposure section of this submission.

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OSHA provides a brief, selective and seemingly irrelevant discussion regarding the possible association between workplace ETS exposures of nonsmoking pregnant women and various adverse reproductive outcomes

OSHA claims that "other reproductive effects that have been ascribed to maternal ETS exposure include miscarriage, an increase in congenital abnormalities, and numerous other physiological effects." (59 FR 15979) As references for this section, OSHA provides citations to the Taylor and Wadsworth (1987) study. (Ex. 8-299) This study examined active maternal smoking and reported no data on ETS exposures during pregnancy. OSHA also cites a response to a Letter to the Editor by Ira Tager. (Ex. 8-297) In this letter, Tager makes no mention of "other physiological effects" in infants born to mothers reportedly exposed to ETS during pregnancy and states the following regarding this issue:

> In discussing the harmful effects of passive smoking [during pregnancy], it is important to point out that there is no body of data that absolutely proves that harmful effects will be experienced.

OSHA also claims that "the effects of environmental [tobacco] smoke exposure on the fetus may have long-term sequelae into childhood and adulthood." (59 FR 15979) The first three studies cited by OSHA make no mention of any purported "effects" of

ETS exposure during pregnancy on the fetus. (Exs. 8-53, 8-181, 8-213) The fourth study cited by OSHA is a study of active maternal smoking during pregnancy. (Ex. 8-225) OSHA also references a CDC survey which provides no information, conclusions, or data regarding the possible effects of maternal ETS exposure during pregnancy. (Ex. 8-51) Finally, OSHA again cites the response to the Letter to the Editor by Ira Tager outlined above. (Ex. 8-297)

OSHA states that exposure to ETS may be "reflected in the growing child as reductions in lung development" and that "this is especially relevant if that child continues to be exposed to ETS throughout childhood and into adulthood." (59 FR 15979) OSHA again references the Tager letter above to support this point. (Ex. 8-297) It also cites a paper which reports:

> Paternal smoking does not appear to be the environmental exposure leading to both childhood respiratory illness and adult chronic air-flow limitation. [emphasis added] (Ex. 8-177)

OSHA DOES NOT PROVIDE A BALANCED AND COMPLETE DISCUSSION OF THE LITERATURE AVAILABLE, I.E., OSHA FAILS TO MENTION STUDIES WHICH REPORT NO ASSOCIATION BETWEEN EXPOSURE TO ETS DURING PREGNANCY AND ADVERSE REPRODUCTIVE OUTCOMES

In 1992, one group of researchers examined the issue of maternal ETS exposure during pregnancy and spontaneous abortion

(miscarriage).[1] Although the authors reported that paternal smoking was not associated with spontaneous abortion (after consideration of other independent risk factors), exposure to ETS from sources other than the father was reportedly associated with an increased risk of miscarriage. This is surprising considering that fact that the authors observed no excess risk of spontaneous abortion among "moderate" smoking mothers or among those who actively smoked an average of more than 20 cigarettes per day in the first trimester. The inconsistent and contradictory data from this study appear to be the result of inadequate control for other factors.

Other research has suggested possible associations between maternal exposure to ETS during pregnancy and infant size, premature labor and neonatal asphyxia.[2] In contrast, Savitz, et al., reported that they could provide "no definitive evidence" of an association between paternal smoking and any birth defect.[3] Other authors similarly have reported no associations between maternal exposure to ETS during pregnancy and the incidence of heart defects,[4-5] premature rupture of amniotic membranes,[6] strabismus (an eye disorder)[7] and various other birth defects.[8]

Although several isolated studies have suggested an association between reported exposures to ETS during pregnancy and various conditions in the infant, the data must be considered both

inconclusive and, in the words of one scientist, "controversial."[9] Another author has suggested:

> Much remains to be done in order to determine whether ETS has any effect on human reproduction or development. Improvements in design of epidemiologic studies of ETS effects, particularly in the areas of exposure assessment and validation and elimination of confounders, are especially important. In addition, appropriate animal studies would be helpful in allowing definitive conclusions to be drawn.[10]

DESPITE THE IRRELEVANCE OF MUCH OF THE LITERATURE OSHA CITES, OSHA STILL FAILS TO PROVIDE A CRITICAL DISCUSSION OF THE PROBLEMS INHERENT IN SUCH EPIDEMIOLOGICAL STUDIES

Probably the most important problems in epidemiologic studies are the lack of quantitative exposure data and the lack of adequate control for potential confounding factors. Exposure estimates in these studies are based on questionnaire information, the validity of which have been questioned repeatedly in the scientific literature.[10] OSHA conveys the impression that confounding factors have been adequately ruled out in these studies. OSHA fails to mention data which, after being adjusted for other factors, have failed to reach statistical significance. The control of confounding variables is of extreme importance in ETS studies examining birth weight. ETS exposure has been shown to "correlate positively with a number of known determinants of

decreased birth weight," including lower socioeconomic status and poor nutrition.[10] Another group of authors suggests that "it is generally believed that birth weight, perinatal mortality and other measures of reproductive health are sensitive markers of social conditions."[11]

OSHA claims that confounding factors have been adequately ruled out as an explanation for the reported association between paternal smoking and reductions in infant birthweight. OSHA mistakenly arrives at this conclusion by taking factors that were controlled for in a few of the studies and generalizing to the entire population of studies.

In addition to these general problems, there are problems specific to the studies OSHA chose to cite in its Proposed Rule. For example, to support its statement that "data on the reproductive effects due to the exposure of nonsmoking pregnant women to ETS has been presented in many studies," OSHA references two abstracts and six papers. (Exs. 3-438, 8-92, 8-174, 8-208, 8-273, 8-285, 8-287, 8-299) A complete review of the statistical procedures and methods, let alone the data, cannot be performed on abstracts.

There are also problems specific to several of the papers cited by OSHA. For example, the Martin & Bracken paper reported

that women who were reportedly exposed to someone else's cigarette smoke for at least two hours per day, either at home or at work, during pregnancy delivered infants that were, on average, 24 grams lighter than the infants of women who were reportedly not exposed to ETS. (Ex. 8-208) This relationship was not statistically significant. This paper is also used to provide support for OSHA's statement that "passive exposure to tobacco smoke is estimated to double the risk of low birthweight in a full-term baby." (59 FR 15979) While the authors reported a relative risk of 2.7 (95% confidence interval 1.05-4.50) for a subgroup of the women, when all women with full-term deliveries were included in the analysis there was no statistically significant relationship between reported exposure to ETS during pregnancy and a reduction in infant birthweight. As one author wrote in 1990:

> The analyses reported by Martin and Bracken (1986) are inconsistent and difficult to interpret. This may be due, at least in part, to the authors' use of a relatively crude estimate of ETS exposure. Their findings would have been more meaningful if further information had been available regarding exposure intensity and duration. Further, since questionnaire data were solicited early in gestation, intensity and duration of ETS exposure may have changed considerably for a number of subjects over the remainder of pregnancy.
>
> Potentially important confounders, such as maternal weight and the child's sex, were apparently not determined during the data collection, and thus were unavailable for the

> analysis, although such variables may significantly influence birth weight.
>
> It is puzzling that the authors used statistical significance as the criterion for inclusion of potential confounders in their multiple regression analyses when, according to various experts, this may eliminate important confounders (e.g., because of low statistical power or multicollinearity).[10]

It should not, then, be surprising that Martin and Bracken conceded that "whether the association found [between decreased infant birthweight and ETS exposure during pregnancy] is due to passive smoking or to some other related factor is unclear" [emphasis added].

The Rubin (1986) paper reported that each pack of cigarettes smoked by the father each day during a mother's pregnancy was associated with a 120 gram reduction in the offspring's birthweight. (Ex. 8-273) However, even a reviewer who is critical of parental smoking, Trichopoulos, has stated that the reported reduction in birthweight reported by Rubin was "extraordinarily large" probably because of poor control for confounding factors.[12] Similarly, another author wrote the following regarding the 1986 Rubin paper:

> An ETS effect of this relative magnitude seems improbable, however, when the relative degrees of exposure are considered. Moreover, in their regression analysis of paternal smoking effects, the authors failed to include interaction terms. This is true even though

> they reported finding a significant interaction between social class and paternal smoking effects, and earlier studies had reported interactions between effects associated with maternal and paternal smoking status. Rubin and coworkers failed to control for a number of other potential confounders as well.[10]

A study which reports the following is also cited by OSHA:

> We report <u>negative results</u> on induction of chromosomal damage in 2 separate groups of intensive involuntary exposure to tobacco smoke, non-smoking restaurant personnel and newborn children of smoking mothers. (Ex. 8-287) [emphasis added]

Finally, the other study cited by OSHA is a paper on the possible effects of active smoking by the mother during pregnancy. (Ex. 8-299) The paper contains no data on pregnant mothers who are exposed to ETS in the workplace during pregnancy.

OSHA fails to discuss a number of other studies which have reported no statistically significant associations between maternal exposure to ETS during pregnancy and the delivery of low birthweight infants.[13-19] In fact, one study considered 57 different risk factors for low birthweight infants and reported that paternal smoking (as an index of exposure to ETS) had no statistically significant effect on infant birth-weight.[13]

OSHA does not provide a detailed discussion of the clinical significance, if any, of the reported decrements in infant birthweight allegedly associated with ETS exposure of the mother during pregnancy.

OSHA'S CLAIM THAT PRENATAL EXPOSURE TO ETS AND EXPOSURE TO ETS AS A CHILD MAY INCREASE AN INDIVIDUAL'S CANCER RISK BY A FACTOR OF TWO IS UNSUPPORTED BY A THOROUGH REVIEW OF THE AVAILABLE LITERATURE

In an attempt to substantiate its claim regarding the possible risk of cancer in nonsmokers exposed either prenatally or as a child to ETS, OSHA cites three papers. (Exs. 8-65, 8-164, 8-252) The first study reported an association between exposure to maternal smoking during childhood and an increase in lung cancer risk in nonsmokers in only one subgroup of subjects. Janerich et al., 1990, reported only one statistically significant risk estimate out of 13 exposure categories. The third reference is a reply to a Letter to the Editor regarding the Pershagen 1987 study (which itself is not referenced by OSHA). (Ex. 8-252) The Pershagen study reported "no consistent evidence of an effect," and the 95% confidence interval encompassed 1.0 for both histologic groups. Therefore, the studies' risk estimates were not statistically significant.

The two studies and the Letter to the Editor cited by OSHA examine the potential effect on an individual's cancer risk from exposure to parental smoking during childhood. These studies are therefore irrelevant to a discussion of potential health effects on the fetus from exposure of the mother to ETS in the workplace during her pregnancy. Nonetheless, a reviewer of this literature concluded that "the data do not indicate any association at all between risk of lung cancer in never smokers and exposure to ETS at work, or in childhood."[20]

OSHA PROVIDES A REFERENCE TO ONE EXPERIMENTAL ANIMAL STUDY WHICH IT CLAIMS IS SUPPORTIVE OF THE CONTENTION THAT EXPOSURE OF PREGNANT MOTHERS TO ETS IN THE WORKPLACE PRESENTS A SIGNIFICANT RISK OF MATERIAL HEALTH IMPAIRMENT TO THE FETUS

Although OSHA acknowledges that "experimental research on the adverse reproductive effects associated with ETS exposure in animals is limited," they do report on one study. (Ex. 8-6) This study reported that sciatic nerve tissue taken from the offspring of fresh sidestream smoke-exposed female mice revealed "definite toxic effects" on the neonatal tissue. There are several limitations of this study. Probably the most important is the small sample size. The authors reported data on only six exposed offspring and six controls. In addition, COHb levels of the exposed pregnant mice were 9%, which the authors report is

"equivalent to that found in humans who actively smoke 10-20 cigarettes per day." As one author suggested, studies which simulate mainstream smoke and employ levels equal to or greater than those expected from active smoking generate data which "are not appropriate for assessment of the likelihood or nature of ETS effects." The author also suggested that the authors of such studies should realize that "careful attention must be given to ensure that observed effects are not simply the result of maternal toxicity and systemic stress resulting from excessive doses."[10]

The authors of this study themselves also noted that these reported changes have been observed in other studies, including studies of the diabetic strain of the C57BL/KsJ mouse and in studies of decreased maternal food intake. They concede that "the irregularities noted in our investigation could be attributed to causes other than cigarette smoke inhalation as previous studies demonstrate."

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SECTION X

MATERIAL IMPAIRMENT: GENOTOXICITY

GENOTOXICITY

OSHA FAILS TO JUSTIFY ITS INCLUSION OF GENOTOXICITY AS A SEPARATE CATEGORY UNDER THE MAJOR HEADING OF "HEALTH EFFECTS"; OSHA FAILS TO SHOW THAT GENOTOXICITY IS A MATERIAL IMPAIRMENT TO HEALTH

Genotoxicity refers to damage to DNA (the hereditary material). Permanent, heritable changes in the DNA are called mutations, and may be examined in tests for mutagenicity, such as the Ames test, in which mutation rates are measured in a particular strain of bacteria. Some correlations have been made between mutagenicity and carcinogenicity (the ability to induce cancer).

Other tests used to assess genotoxicity include the sister chromatid exchange (SCE) assay, which measures the frequency of exchange of portions of genetic material between two identical strands of DNA, and the chromosome aberration (CA) assay, which measures structural irregularities in strands of DNA. DNA adducts, abnormal configurations or clumping of the genetic material, may also be examined.

All forms of life are constantly exposed to physical and chemical agents in the environment (e.g., radiation) and to endogenous (internal) agents with the ability to cause changes in DNA. DNA has been called an "unstable" molecule, and it has been

noted that endogenous DNA damage may occur at the rate of 100,000 base pairs per cell, per day.[1,2]

According to Bruce Ames, developer of the Ames assay for mutagenicity, human exposure to potentially mutagenic or carcinogenic substances is much greater than generally appreciated, e.g., the environment can be thought of as "filled with potential carcinogens."[2]

Moreover, conclusions about genotoxicity obtained from in vitro systems, while certainly providing some information about the substance being tested, must nevertheless be put into the proper biological context. The magnitude of a genotoxic response in the whole organism may be substantially different than that observed in a bioassay. As Ames and Gold noted[3]:

> [H]umans have numerous inducible defense systems against mutagenic carcinogens, such as DNA repair, antioxidant defenses, glutathione transferases, and so forth . . . [L]ow doses of carcinogens appear to be both much more common and less hazardous than is generally thought.

Given the ubiquity of mutagens in the environment and the existence of numerous "defense systems" against mutagens, OSHA has not provided sufficient information in the Proposed Rule to support

its treatment of genotoxicity as a "health effect" or to establish genotoxicity as a material impairment to health.

OSHA'S REVIEW OF THE LITERATURE ON THE CLAIMED GENOTOXICITY OF ETS IS INCOMPLETE; A REVIEW OF ADDITIONAL RELEVANT STUDIES PROVIDES NO SUPPORT FOR OSHA'S CLAIM

OSHA's discussion of genotoxicity begins with a series of statements about the claimed correlations between genotoxicity and carcinogenicity. (Section II.C.7., 59 FR 15981) OSHA fails to provide references in support of these statements, although they appear to be OSHA's justification for discussing genotoxicity.

The Proposed Rule then contains a discussion of a number of studies in which cigarette smoke or cigarette smoke condensate was tested in the Ames Salmonella typhimurium assay, and an increased mutation rate was reported. OSHA's inclusion of studies dealing with mainstream and sidestream smoke again reveals the misconception pervading the Proposed Rule that ETS, mainstream, and sidestream smoke are equivalent.

OSHA omitted at least one relevant study from this discussion in the Proposed Rule. In 1991, Bombick, et al., reported on a cellular smoke exposure techniques using rat liver cells and the Ames Salmonella assay. After a three-hour exposure

using ETS at a concentration of 1.5 mg total particulate matter/m^3, the authors report[4]:

> Using the neutral red cytotoxicity and Ames mutagenesis assays there were no differences observed in the ETS-exposed cells and their respective room air controls, indicating that ETS was biologically inactive as tested. [emphasis added]

The Proposed Rule also discusses studies reporting that various constituents and extracts of ETS collected from indoor air are capable of inducing mutations in the Ames assay are also discussed. (Exs. 4-202, 4-5, 4-198, 4-201, 4-203) However, the significance of such reported findings has not been established. Virtually all air samples, whether in the presence or absence of smoking, can be shown to be mutagenic in various bioassays. Indeed, no substance, including foods and natural materials, has been unequivocally shown to be free of mutagenic and/or carcinogenic properties.

Of relevance, a study not cited by OSHA has reported that sidestream smoke exhibits reduced activity as it ages and becomes diluted, that is, as it becomes ETS.[5] Sonnenfeld and Wilson report on an experiment in which cultured mouse fibroblast-like cells were exposed to mainstream or sidestream smoke of various ages. In

this report, cytotoxicity (cell mortality) is used as a measurement of DNA damage sufficient to cause cell death. The authors write:

> Aging of SS smoke resulted in a rapid decline in the mortality generated by the smoke. As calculated from the linear regression curve, an increase in age of SS smoke of 30 [seconds] after generation would have resulted in a total loss of cytotoxic effects. [emphasis added]

The Proposed Rule almost completely overlooks genotoxicity studies using bodily fluids of persons exposed to ETS; these data do not allow a conclusion about genotoxicity to be reached

One area of research essentially overlooked in OSHA's Proposed Rule comprises those studies that have compared the mutagenicity of body fluids of nonsmokers exposed to ETS and nonsmokers not exposed to ETS. Several of these studies report no significant difference in mutagenic activity.[6-10] (Ex. 8-152)

For instance, in research by a team of German researchers, ten nonsmokers were exposed to ETS, generated by human smokers, for eight hours under two exposure conditions.[9] The two experiments were characterized by CO levels of 10 ppm and 25 ppm, respectively; according to the authors, both exposure regimes represent higher exposures than "real-life" situations. Elsewhere,

they described Experiment 2 as "far from being realistic,"[6] and bearing "no relation to a real-life situation."[8] In addition, the authors controlled for the effect of mutagens from the diet by keeping their subjects on a diet low in polycyclic aromatic hydrocarbons. Urine samples from both smokers and nonsmokers were tested in the Ames Salmonella assay. The authors report:[9]

> All urine extracts of ETS exposed non-smokers were found to be negative in the mutagenicity test when applying the [criterion] of Ames (doubling of spontaneous mutation rate).

Thus, even at exposure levels higher than would be expected on average, no increase in mutagenicity could be measured. These data do not support claims that ETS exposure is associated with an increase in mutagenic activity.

Citing the high variability of measures of urinary mutagenicity and questions about the relevance of increased urinary mutagenicity to cancer risk, the authors write:[8]

> These considerations lead to the conclusion that measuring the urinary mutagenic activity, at least in passive smokers, is not an appropriate method of predicting an increased risk to human health.

The authors also say:[8]

> The data suggest that nonsmokers in real-life situations take up very low doses of ETS constituents, and detoxification of the genotoxic substances inhaled is effective.

And:[10]

> Whether ETS exposure can lead to an elevated urinary mutagenicity is a matter of controversy. In most investigations no significant increase has been observed. . . .
>
> In our experience, the Ames test for detection of urinary mutagenicity is far too insensitive to assess such a low exposure. The results of our investigations, as well as those of other authors, suggest that urinary mutagenicity, which would be a potential marker for ETS particle exposure, remains unchanged after ETS exposure.

The few studies reporting statistically significant increases in urinary mutagenicity among individuals exposed to ETS did not employ realistic levels of exposure to ETS, and they did not control adequately for the presence of mutagens in the diet of the study subjects.[11-13] For instance, in the Bos, et al., study, the exposure condition consisted of the smoking of 157 cigarettes over six hours in a room with "poor ventilation."[11] The relevance of such an exposure to "real-life" conditions is certainly questionable. With respect to diet, Bartsch, et al., acknowledge, concerning their study, that[13]

Urinary mutagenicity is influenced also by dietary habits; although we collected information on diet, the dimension of the study (particularly as far as passive smokers are concerned) does not allow adequate statistical treatment of this potential confounding factor.

Other related studies have examined levels of various DNA changes in nonsmokers exposed to ETS.[1,14-17] (Exs. 8-152, 8-287) Based on the data presented in these studies, nonsmokers exposed to ETS do not appear to exhibit increased DNA adduct formation, nor do studies report increased levels of chromosomal changes in cells of nonsmokers exposed to ETS. Discussion of these studies follows.

Collman, et al., collected data from 16 nonsmokers, 15 "passive smokers" (currently living with one or more smokers), and 13 current smokers, all women.[14] Sister-chromatid exchange (SCE) frequencies in lymphocytes (a type of white blood cell) were compared with and without coincubation with a chemical that enhanced the frequency of SCEs. Based on both assays, the authors report that "the frequency of SCEs in persons passively exposed to smoke was not higher than in nonsmokers."

In a report by Husgafvel-Pursiainen, peripheral blood lymphocytes were examined for SCE frequency.[15] Study groups consisted of 12 smoking waiters and waitresses, 20 nonsmoking waiters and waitresses who were occupationally exposed to ETS, and

14 nonexposed office workers. The author reports that "[t]he mean SCE level in exposed non-smokers did not differ from that observed in the non-exposed group." Although no ETS measurements from the restaurants were reported, the author characterizes them as "heavily polluted," and the exposure as "long-term." This study, which reports data from persons exposed in a "real-life" situation, does not support claims of the genotoxicity of ETS.

Chromosomal aberrations (CAs) and SCEs were examined in peripheral blood lymphocytes from nine smoking waiters, 16 nonsmoking waiters exposed to ETS at work, and 7 reportedly nonexposed nonsmokers by Sorsa, et al. (Ex. 8-287) The authors report that "[n]o significant differences were seen between the groups or subgroups in the 2 parameters." Thus, no "genotoxic" effects could be detected in persons exposed to ETS at "real-world" levels.

Holz, et al., report that DNA adduct levels were compared in monocytes (a type of white blood cell) of smokers and "heavily exposed passive smokers," who had been exposed in a chamber.[16] DNA adducts above background were reported in smokers; they disappeared in less than 40 hours. The authors report no above-background adduct levels in study subjects exposed to ETS.

In a study by Gorgels, et al., 50 self-reported ETS-exposed men ("passive smokers"; average 72.8 hours exposure per week) were compared with 56 self-reported low ETS-exposed men (average 5.1 hours per week).[17] SCEs in cultured lymphocytes were examined; the authors reported that "[n]o difference was observed between low exposed non-smokers and the passive smokers." They concluded:

> Our results are in accordance with previous smaller studies in less homogeneous populations of non-smokers. These studies also failed to demonstrate even a tendency for an association between passive smoking and SCE levels. . . .

Five male smokers, five male nonsmokers, and five male nonexposed nonsmokers were compared in Holz and colleagues' 1993 paper.[1] The endpoint examined was DNA single-strand breaks (SSBs), "considered to be an important parameter of genotoxic stress," in lymphocytes. The authors write:

> All probands revealed measurable and varying SSB levels. Since DNA is an unstable molecule and estimated endogenous damage exceeds 100000 affected base pairs per cell per day, we assume that SSB base levels reflect unrepaired lesions. Active smoking caused an increase in SSBs in peripheral blood lymphocytes. This effect could not be found in passive smokers . . .

ETS exposure in this study consisted of five smokers each smoking 24 cigarettes in eight hours in an exposure chamber. This study provides no support for claimed genotoxic effects of ETS, even at a high exposure level.

Conclusion

This review of data from studies in which genotoxicity was assessed in persons actually exposed to ETS thus provides little, if any, support for the contention that ETS is genotoxic at levels encountered in workplaces and other indoor environments.

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SECTION XI

THE PROPOSED RULE ON ETS:

FEASIBILITY AND ALTERNATIVES

OSHA'S PROPOSED RULE ON ETS: FEASIBILITY AND ALTERNATIVES

OSHA HAS NOT DEMONSTRATED ANY CONTRIBUTION OF ETS TO CONSTITUENT LEVELS ABOVE BACKGROUND LEVELS IN THE WORKPLACE; OSHA HAS NOT MADE A DETERMINATION OF "SIGNIFICANT RISK" FOR AMBIENT ETS EXPOSURE LEVELS; OSHA FAILS TO PROVE THAT ETS CANNOT BE ADDRESSED THROUGH PROPER VENTILATION; OSHA FAILS TO EXPLORE ALTERNATIVES TO SMOKING BANS

OSHA states that the "primary objective of the tobacco smoke provision is to eliminate the nonsmoker's exposure to ETS. Under the Proposed Rule, firms will have the option of either banning smoking of tobacco products or permitting smoking only in designated areas." (59 FR 16016) The designated smoking area must be completely enclosed with a separate exhaust directly to the outside. In addition, the area must be negatively pressurized to prohibit exposure of any ETS constituent outside the designated area. (59 FR 16029) The Proposed Rule on ETS, according to OSHA, reduces "significant risk of material health impairment to the extent feasible." (59 FR 16013)

The Proposed Rule, however, does not explain why the complete elimination of ETS is required or how the studies that OSHA selected for its analysis of significant risk warrant the complete elimination of ETS. According to a recent court opinion (AFL-CIO v. OSHA, 965 F.2d 962, *975), OSHA's determination that a

new standard is "reasonably necessary or appropriate" and that it "most adequately assures . . . that no employee will suffer material impairment of health or functional capacity, . . . necessarily requires some assessment of the level at which significant risk of harm is eliminated or substantially reduced." OSHA does not provide an assessment of the level of "significant risk of harm." That is because its analysis of significant risk for ETS is based upon two epidemiologic studies that are inconsistent with the body of evidence on health effects from workplace exposures to ETS, and from which estimated risks are generated without reference to actual ambient measures of ETS exposures.

OSHA provides no reason for setting its ETS standard at "zero" exposure. The zero level of exposure does not follow from OSHA's analysis of significant risk. It does not follow from OSHA's contention that its ventilation-based IAQ standard will not be effective in the removal and dilution of ETS constituents because "air exchange rates in non-industrial workplaces are not designed to control the risks of ETS exposure" (59 FR 15991), or because "the carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant." (59 FR 15992) As discussed in Section IV of this submission, ordinary air in public places and workplaces contains many of the same substances imputed to ETS, in the complete absence of smoking

Such substances, some of which are designated as "carcinogens" by OSHA, are produced by many common sources, and complete removal of ETS from the ambient air will not eliminate exposures to them. Moreover, some individual constituents found in both indoor air and ETS are already regulated by OSHA. OSHA establishes permissible exposure levels to such airborne substances presumably at levels protective of employee health and at which no significant risk of material impairment exists. The levels of exposure are typically far greater than any actually generated by ETS. OSHA's own Proposed Rule on IAQ does not call for the complete elimination of such substances, some of which are "carcinogens," but for the reduction of exposures to an "acceptable" level by ventilation.

OSHA thus has not demonstrated the extent to which ETS contributes to constituent levels above background levels in the workplace, it has not made a determination of "significant risk" for ambient ETS exposure levels, and it fails to prove that ETS cannot be addressed through proper ventilation, as spelled out in its own IAQ standard.

While the complete elimination of ETS is achievable through a smoking ban,* the complete elimination of the same

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*. OSHA's rulemaking on ETS is directed at stopping the smoker rather than "protecting" the nonsmoker. A smoking ban with the express purpose of altering smoking behavior may be seen

(continued...)

constituents from other sources is not possible. If a comprehensive indoor air quality standard were truly the goal of the Proposed Rule on IAQ and ETS, then OSHA would have explored alternatives that are functionally equivalent in effectiveness to smoking bans in the reduction of exposures to ETS -- alternatives that would not seek to alter the behavior of the smoking workforce. A discussion of alternatives to smoking bans and/or separately ventilated smoking rooms follows below.

Current design and operation criteria for ventilation provide for the effective dilution and removal of ETS constituents from workplaces in which smoking is unrestricted; a ventilation Standard, ASHRAE 62-1989, has been incorporated into the major building codes in the U.S.; since 1990, the Standard has provided design and operations criteria for ventilation systems in new, remodeled and renovated buildings; the Standard has been incorporated by reference into OSHA's Proposed Rule on IAQ

The Proposed Rule states, without justification, that ventilation cannot be used to address the "risks" purportedly associated with exposures to ETS. The Proposed Rule specifically cites the "failure" of ASHRAE Standard 62-1989 in the "elimination" of "risks" from ETS exposures. (59 FR 15992) While ASHRAE Standard 62-1989 does not explicitly address purported "risks" from indoor air exposures, it establishes ventilation rates for various

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*.(...continued)
as an attempt at "social engineering" and clearly is not, and ought not to be, within OSHA's regulatory framework.

locations in order to "control carbon dioxide and other contaminants with an adequate margin of safety, and to account for variations among people, varied activity levels, and a moderate amount of smoking" [emphasis added].[1] The Chairman of the ASHRAE Standards Project Committee for Standard 62-1989, Mr. John Janssen, writes:

> Laboratory research at Yale University and at the Technical University of Denmark has shown that 15 cfm of outdoor air will dilute occupant odors to a level acceptable to at least 80 percent of the people entering an occupied space from outdoors. Research on tobacco smoke odor at Yale's John B. Pierce Laboratory has also shown that with today's reduced smoking rate 15 cfm of outdoor air will dilute environmental tobacco smoke to a level acceptable to 70 percent of the people entering an occupied space where about 27 percent of the occupants smoke at a rate of 1.25 cigarettes per hour. This equates to one pack per 16 hour day. Other calculations on the tobacco smoke perception of nonsmoking occupants in a room for 15 minutes with smokers show that nonsmoking occupants will register 90 percent acceptance under the assumed conditions. Thus, Standard 62-1989 appears to be able to control tobacco smoke odor under minimum smoking conditions.[2]

The minimum ventilation rate recommended by ASHRAE Standard 62-1989, 15 cfm outside air/person, is the level at which odors, metabolic byproducts, and ETS are effectively diluted and removed.[3-5] (Ex. 3-440) The effectiveness of the minimum ventilation rate for the dilution of ETS has been evaluated by scientists.[6-8] In 1990, Pedelty and Holcomb demonstrated that air

quality in areas where smoking is permitted ad lib does not differ significantly from the quality of air in nonsmoking areas, where both areas are supplied with the minimum outdoor air ventilation rates recommended in ASHRAE 62-1989.[6]

In their review of ETS-related air quality monitoring in different workplaces under various smoking conditions, researchers from TDSA Ltd. conclude "in office areas in which (a) smoking is allowed and (b) outside air ventilation rates meet or exceed the ASHRAE Ventilation Standard, nicotine concentrations have typically been less than 5 ug/m^3 and respirable suspended particles have ranged between 20 ug/m^3 and 60 ug/m^3.[7] (Ex. 3-1073)

In their submission to the OSHA RFI Docket, scientists summarized the results of their paper on the measurement of ETS in 585 offices. (Ex. 3-1053) The authors write:

> Computer analysis shows that when "blind-folded" for presence or absence of smokers, in most cases realistic smoking levels do not significantly influence the aspects of air quality that were measured, and spillover from smoking areas into nonsmoking areas appears to be minimal. This work further reinforces the position the American Society of Heating, Refrigerating and Air-Conditioning Engineers has taken on ETS in office buildings in ASHRAE Standard 62-1989, that acceptable air quality can be maintained in properly ventilated offices with a moderate amount of smoking even without smoker segregation.

Professor Alan Hedge offers the following observation on the basis of his extensive experience in monitoring ETS constituents during investigations of sick-building syndrome: "Our data show that modern ventilation systems are capable of diluting the small pollutant loads from smoking at the levels which we observe, without necessarily exposing nonsmokers to significantly elevated levels of indoor air pollutants." (Ex. 3-955)

Company scientists from R.J. Reynolds (RJR) reported on a recently completed study of four office buildings.[8] (Ex. 3-1087) Two of the buildings investigated had a policy of unrestricted smoking and, in two other buildings, smoking was restricted to separately-exhausted lounges. Regardless of smoking policy, RJR reports that ventilation and indoor air quality indicators were "well within applicable standards." The authors write:

> In summary this study demonstrates conclusively (a) that with a HVAC system that is adequately designed, operated in accordance with current ASHRAE standards and properly maintained, all indicators for ETS are at extremely low, de minimis levels, even in the presence of substantial smoking activity, and (b) that such smoking activity has a negligible effect on contaminant levels in buildings where smoking is unrestricted.

They conclude:

> RJR believes, based on its own detailed research and the consistent results of other workplace assessments, that a properly designed and maintained HVAC system that is

operated in accordance with the ventilation rate procedures of the ASHRAE Standard 62-1989, will be effective in assuring that exposures to ETS will be de minimis.

Thus, in buildings meeting the ventilation rates specified in ASHRAE 62-1989, return air from areas in which smoking is permitted will be diluted by outside air, and the mixture of return and outside air will be filtered prior to returning to the supply system. The dilution factor accounts for the low levels of ETS constituents measured in nonsmoking areas, as documented in the above studies.

Simple physical separation of smokers and nonsmokers has been effective in the reduction of nonsmoker exposure to ETS; simple spatial separation of smokers and nonsmokers, even under conditions of recirculated ventilation, effectively minimizes ETS exposure for nonsmokers; data do not support a significant reduction in ETS exposures beyond adequate ventilation and/or simple separation of smokers and nonsmokers

Scientific studies indicate that the simple physical separation of smokers and nonsmokers, even under a shared ventilation system with recirculated air, can effectively minimize nonsmoker exposures to ETS.[7,9-20] Other studies indicate that smoking bans and/or separately ventilated smoking areas do not significantly reduce ETS exposures beyond reductions achieved through simple separation and/or adequate ventilation.[8,9,12-14,17]

A 1990 study by Vaughn and Hammond, cited in the Proposed Rule (59 FR 15991), examined the impact of smoking policies on ETS constituent levels in a high-rise building.[9] The authors reported an 80 percent reduction in exposure to ETS constituents in nonsmoking workareas after designation of a smoking area on a floor that utilized a common (recirculating) air-handling system. Exposure levels to nicotine in nonsmoking workareas prior to the designation of the smoking area were 2.0 ug/m^3; after institution of the smoking policy, nicotine levels dropped to 0.1-0.3 ug/m^3. A complete smoking ban on another floor in the building produced a 95+ percent reduction in ETS constituents, only a 15 percent exposure reduction beyond simple separation of smokers and nonsmokers.[9]

Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable.[10] Smoking was restricted to designated areas with local air filtration systems. The authors concluded that spatial restrictions are "effective in minimizing the impact of environmental tobacco smoke on indoor air quality."[10]

In a similar study, Canadian researchers compared measured ETS constituents in offices where smoking was regulated and unregulated.[7,11] They reported no significant differences in average ETS constituent levels between nonsmoking offices that

received recirculated air from designated smoking areas, and nonsmoking offices that did not receive recirculated air. Nicotine concentrations reported for nonsmoking areas were only marginally above limits of detection and quantitation; there were no measurable differences in particles or carbon monoxide levels in nonsmoking areas that did or did not receive recirculated air from smoking areas. The researchers concluded:

> The results indicate that the provision of a designated, but not separately ventilated, smoking area can effectively eliminate or drastically reduce most components of environmental tobacco smoke for nonsmoking offices.[11]

In 1991, Hedge, et al. examined the effects of smoking policies on indoor air quality in 18 private-sector buildings.[12] The study covered over 3,000 workers. They concluded:

> Comparison of all open-office sites between policies showed no significant differences in levels of carbon monoxide, carbon dioxide, formaldehyde or respirable particulates. . . . Smoking policy had a relatively small effect on IAQ for the pollutants measured. For most of these pollutants, there were no significant differences in concentrations among offices in SP (smoking-prohibited) buildings, nonsmoking office areas in RF (smoking restricted to rooms with local filtration), RSV (smoking restricted to rooms with separate ventilation) and RMP (smoking restricted to rooms with no location air treatment) buildings, and office areas in RWS (smoking restricted to open-plan cubicle workstations and enclosed office) buildings. There was a significant effect of smoking policy on UVPM and formaldehyde in these office areas . . . however, all

concentrations of UVPM and formaldehyde were low.

A 1993 follow-up study by the same authors compared ETS constituent levels in 27 office buildings under five different kinds of smoking policies.[13] The smoking policies ranged from unrestricted smoking to the complete prohibition of smoking. The authors report that nicotine and tobacco-specific particles (UVPM-RSP) were measurable in offices that permitted smoking, but exposures to other airborne substances were similar across all buildings, regardless of smoking policy. The authors estimated that a typical nonsmoker would be exposed to the nicotine equivalent of approximately three cigarettes per year in open-plan offices with smoking restricted to enclosed parameter offices. Simple separation of smokers and nonsmokers under a common ventilation system was estimated to result in nicotine exposure levels of no more than five cigarette equivalents per year.

A 1993 Canadian study compared exposures to ETS constituents in three buildings before and after smoking bans.[14] The authors reported a significant reduction in average levels of volatile organic compounds in the buildings after the smoking ban, a result they could not explain and one that is inconsistent with other studies that demonstrate no significant contributions from ETS to indoor levels of total volatile organic compounds.[15,16] The smoking ban, however, had no significant effect on overall

exposures to carbon monoxide or particles, or on cotinine levels in body fluids of nonsmokers.

Similarly, Proctor (1987) monitored ETS constituents before and after a smoking ban on public transportation in the United Kingdom.[17] While nicotine concentrations decreased from 7 ug/m^3 to 3 ug/m^3 in nonsmoking compartments after the ban, particulate and carbon monoxide levels remained unchanged. This suggests that ETS contributions to levels of particulate and CO in public transportation are not significant.

In another study by Proctor and co-workers (1989), the researchers measured nicotine, RSP, carbon monoxide, carbon dioxide, and volatile organic compounds in the air of smokers' and nonsmokers' offices.[15] The data indicate little nonsmoker exposure to various ETS constituents through simple separation. The average UVPM-RSP level in nonsmokers' offices was 8.8 ug/m^3; the median nicotine value was less than 1 ug/m^3. Carbon monoxide and carbon dioxide levels did not differ appreciably between smokers' and nonsmokers' offices. Overall, levels of volatile organic compounds did not differ significantly between smokers' and nonsmokers' offices.

Bayer and Black (1987) reached a similar conclusion in their investigation of volatile organic compound levels in smokers'

and nonsmokers' offices.[16] They noted that although differences in nicotine concentrations were measurable for offices of smokers compared with nonsmokers, no significant differences in volatile organic compounds were discerned in smokers' and nonsmokers' offices. The researchers observed that "it was not possible" to correlate volatile organic compounds with ETS or to attribute the source of various volatile organics to ETS.

A 1989 study performed for the U.S. Department of Transportation on ETS constituent levels aboard commercial aircraft indicates the overall effectiveness of simple separation of smokers and nonsmokers in the minimization of ETS exposures.[18] This study is cited in the Proposed Rule. (59 FR 15991) The researchers reported an average level of 0.11 ug/m^3 nicotine in nonsmoking sections for their sample of 61 domestic commercial flights. The average level was over 100 times lower than that measured in smoking sections; it is equivalent to 1/8000 the nicotine delivery of a single cigarette.

In the largest study of its kind, researchers reported measurements of ETS constituents in 585 offices, many of which were conventional office settings with simple separation of smokers and nonsmokers under common air-handling systems and recirculated air.[19] The researchers concluded that: "[I]n most cases realistic smoking levels do not significantly influence the aspects of air quality

that were measured, and spill-over from smoking areas to nonsmoking areas appears to be minimal." "Spill-over" of tobacco smoke constituents was reported in only four percent of the nonsmoking areas.

Lambert, et al. (1993) examined differences in nicotine and RSP levels in the nonsmoking and smoking sections of restaurants.[20] Simple separation of smokers and nonsmokers in restaurants resulted in substantial reductions of exposure to RSP and nicotine for nonsmokers. Nicotine concentrations averaged 65 percent lower in nonsmoking sections than in smoking sections; RSP concentrations were 40 percent lower. The average concentration of nicotine in smoking areas was 3.2 ug/m^3 compared with 1.0 ug/m^3 in nonsmoking areas. The difference between average RSP levels in smoking and nonsmoking sections was 26 ug/m^3, a level consistent with those reported for the contribution of ETS in homes and in offices with smokers. (See Table V, Section IV)

The studies reviewed above contain data regarding the low levels of ETS constituents in nonsmoking areas under conditions of simple separation of smokers and nonsmokers with recirculation of ventilation air. Data reported in those studies indicate that ETS constituent levels in nonsmoking areas in buildings where smoking is permitted are often only slightly above limits of detection and quantitation, and often statistically indistinguishable from

"background" levels of such constituents found in buildings in which smoking is altogether prohibited. The data support the contention that simple physical separation of smokers and nonsmokers effectively reduces and minimizes ETS exposure in nonsmoking areas, even under conditions of recirculated ventilation.

There are substantial data, submitted to the OSHA RFI docket on IAQ and reviewed in this section, that indicate that typical workplace exposures to ETS constituents are low and reducible to de minimis levels through the simple physical separation of smokers and nonsmokers in conjunction with the current ventilation rates adopted in OSHA's Proposed Rule on IAQ. **The Proposed Rule provides no discussion or scientific data that would support a finding that ETS is related to any material health impairment at exposure levels encountered through simple separation and/or adequate ventilation.**

Negative air pressure zones; if physical grouping of smokers and nonsmokers in discrete areas is desired by employers, prevailing air circulation currents and routes of supply and exhaust air should be considered; if possible, smoking areas should be placed near existing exhausts so that air movement will be directed from nonsmoking to smoking areas, thereby minimizing possible migration of tobacco smoke from smoking areas

According to Hayward, et al. (1993), the effectiveness of a designated smoking area for controlling exposure to ETS in nonsmoking areas is determined by two basic factors.[21] The first requires the successful dilution and removal of ETS constituents within the smoking area. The ventilation rate, either through outside air or transfer air (the air from other zones within the building), is the most critical determinant for the dilution and removal of ETS constituents. Outside air should be supplied at rates designated by ASHRAE Standard 62-1989.

A second factor related to the movement of ETS constituents from smoking areas to nonsmoking areas depends upon airflows within the structure.[21] Airflow is affected by the existence of physical barriers such as walls or partitions, as well as by air pressure relationships within the building. For a designated smoking area without partitions, the simple location of smokers near existing exhausts and the designation of nonsmoking areas near supply air diffusers will prevent movement of ETS constituents into nonsmoking areas. Airflow will be directed from

the nonsmoking or supply air areas into smoking areas, thereby preventing the air from the smoking area from re-entering that of the nonsmoking area. This technique, known as "air pressure zoning," has been described in a recent publication for design engineers:

> Air generally flows from areas of higher air pressure to areas of lower air pressure, from positive pressure in the direction of negative pressure. Using this simple concept, areas set aside for nonsmokers can be maintained at a slight relative positive pressure, while areas set aside for smokers can be maintained at a slight negative relative air pressure. This will produce a slight airflow from the nonsmoking area into the smoking area, keeping the air from the smoking area from mixing with that of the nonsmoking area. Through thoughtful planning and carefully supervised and tested balancing of the HVAC system, the preferences of both smokers and nonsmokers can be accommodated without any additional cost to building operations.[22]

Air pressure zoning involves the use of existing ventilation systems, e.g., supply and exhausts within a building, and will not influence capital costs or operating efficiencies for a building.

An alternative to the separate smoking area required in the Proposed Rule (i.e., an enclosed space with dedicated exhaust under negative pressure) utilizes the theory of negative pressure and the use of transfer air, which is air drawn directly from other parts of an occupied space. The use of transfer air in a practical

smoking lounge design is permitted under ASHRAE Standard 62-1989. The lounge would be ventilated in a way similar to the way restrooms are ventilated and exhausted. Restrooms in public buildings are equipped with exhaust ventilation for the removal of odors, etc. The restroom often draws its supply air from adjacent areas such as corridors that are close to the restroom. The rooms adjacent to the restroom are not fitted with comparable exhaust capabilities. The exhaust air from the restroom creates a negative pressure relative to its adjoining areas. Air is thus "transferred" from adjacent areas of positive pressure into the restrooms and, if the exhaust is working properly, the result is the creation of a negative pressure zone.

The 1993 publication by Hayward, et al. examined the effect of negative pressurization on movement of ETS constituents in three separate buildings.[21] In one of the buildings, the use of a small exhaust fan in the smoking area dramatically reduced migration of ETS constituents into nonsmoking areas. Exposures to nicotine and RSP in nonsmoking areas were reduced below the limit of quantitation. In a second building, the effects of negative pressurization were less dramatic because the ventilation was very effective from the outset in removing and diluting ETS constituents. A third building was not negatively pressurized in smoking areas and nonsmoker exposure to ETS constituents was greater than in the other two buildings.

In 1993, Light and Gay measured nicotine levels in two office buildings with a variety of areas designated for smoking.[23] Nicotine was below the level of detection (less than 0.7 ug/m^3) in most of the sites measured. The authors concluded: "Within the sensitivity of the tests and observations performed, exposure was not documented from the recirculation of air even though many smoking areas were not exhausted to the outside. This suggests that there was little, if any, hazard under the conditions evaluated in areas potentially receiving recirculated ETS but not immediately adjacent to smoking." They reported that "positive pressurization of smoking rooms" led to "intermittent nonsmoker exposure in immediately adjoining areas." If the smoking areas were negatively pressurized, no detectable exposure to ETS constituents occurred adjacent to smoking areas.

Feasibility of OSHA's Proposed Rule on ETS

According to OSHA, "the primary objective of the tobacco smoke provision [of the Proposed Rule] is to eliminate the nonsmoker's exposure to ETS." (59 FR 16016) OSHA further states: "Under the Proposed Rule, firms will have the option of either banning smoking of tobacco products or permitting smoking only in designated areas." (59 FR. 16016) The Proposed Rule requires that designated smoking areas be enclosed, exhausted directly to the outside and maintained under negative pressure. (59 FR 16032)

Under OSHA's Proposed Rule for ETS, constituents imputed to ETS will be "eliminated" from the workplace, while exposures to the same constituents from other sources will be minimized to presumably acceptable levels by the ventilation-based Proposed Rule for IAQ. The foregoing analysis clearly demonstrates that: (1) ETS constituent levels in typical workplaces are low and nonsmoker exposure to ETS constituents is minimal; (2) simple physical separation of smokers and nonsmokers in the workplace provides for significant reductions of already minimal exposures to ETS constituents; (3) adequate ventilation effectively dilutes and removes ETS constituent levels to the extent that levels will often fall below levels of detection or quantitation and will not differ significantly from background levels of constituents generated by other sources; and (4) the negative pressurization of smoking areas will prevent "migration" or "spillover" of ETS constituents into nonsmoking areas.

OSHA's proposal to completely eliminate ETS constituents is a regulation that seeks to modify already insignificant levels of ETS exposure. The alternatives described above were not considered by OSHA, yet they are equivalent in effectiveness to OSHA's proposed requirement of a separately enclosed, separately exhausted, and negatively pressurized smoking room. **The Proposed Rule will produce only trivial and insignificant reductions in**

exposures to ETS constituents over the alternative provisions specified above.

OSHA's Proposed Rule on ETS ostensibly provides a choice regarding smoking for the nation's employers: either ban smoking or construct special smoking rooms. However, the choice is not real. The Proposed Rule constitutes a de facto ban on smoking because OSHA trivializes and minimizes the economic and technological feasibility of providing separate rooms for smoking employees. The "option" of providing a separately ventilated smoking room lies with employers (not building owners), even when the employer leases space for his or her business. OSHA clearly recognizes this impossible scenario in its Proposed Rule, e.g., "since changes in building ventilation systems will be made by landlords, employers may have to negotiate agreements to ensure that they can meet the OSHA Standard. On the requirement for ETS, landlords in turn are likely to pressure employers to ban smoking; thereby forestalling any need for construction of designated smoking rooms." (59 FR 16013)

OSHA declares that problems concerning the technological feasibility of the Proposed Rule "are not evident." (59 FR 16013, 16023) However, the isolation of smokers in a separate room as required by OSHA's Proposed Rule places additional demands on an existing ventilation system. The Proposed Rule's requirement of a

separate exhaust leading directly to the outdoors is not feasible in many buildings.[22] Few existing buildings, particularly high-rise buildings, are amenable to providing exhaust directly to the outdoors from any given location within the building. The option is not technologically feasible in these instances, and the employer, under OSHA's Proposed Rule, would have no choice but to completely ban smoking. The Proposed Rule concedes: "OSHA recognizes that not all establishments will make available designated smoking areas as there may be physical constraints on the option of providing separate ventilation. Such constraints are imposed by the building's design, the building's mechanical ventilation system's capabilities, by cost involved in providing adequate ventilation, by the occupant use of the building." (59 FR 16016) The U.S. EPA recently conceded that "the structural features of many existing buildings make it infeasible or cost prohibitive to construct a smoking lounge" similar to that envisioned by OSHA.[24] The EPA report suggests that smoking lounges would be feasible in only 10 to 20% of the existing buildings in the U.S. Tenants in 80 percent or more of existing buildings would be forced to ban smoking altogether under OSHA's Proposed Rule.

OSHA has placed the burden of its Proposed Rule on ETS upon the nation's employers by presenting them with a "choice" over which they are not empowered (i.e., to ban smoking or restrict it to a specially ventilated room). For the employer who is not also

a building owner, there is no real choice in the matter. For either the tenant or owner of a multi-story building, there may be no "choice" in the matter due to feasibility restrictions. For the small business owner who must lease additional space for a smoking lounge, there may be no "choice" in the matter. For other businesses, retrofit requirements for the construction of an enclosed, separately exhausted and negatively pressurized smoking room may be cost-prohibitive, and the "choice" in the matter disappears. The Proposed Rule does not address these issues.

The minimum outdoor air ventilation rates required in OSHA's Proposed Rule on IAQ are based on versions of ASHRAE Standard 62 (for ventilation); the current ventilation standard, ASHRAE 62-1989, provides minimum outdoor air ventilation rates for ETS; the precursor standard, 62-1973, specified recommended ventilation rates comparable to ASHRAE 62-1989 and served as the ventilation basis for building codes since 1973; OSHA's ventilation recommendation for IAQ will therefore adequately address ETS; OSHA's separation of ETS from general IAQ has no basis

OSHA's Proposed Rule for IAQ (to the exclusion of ETS) states that "employers [must] maintain and operate the HVAC system to provide at least the minimum outdoor air ventilation rate, based on actual occupancy, required by the applicable building code, mechanical code, or ventilation code in effect at the time the facility was constructed, renovated, or remodeled, whichever was most recent." (59 FR 16026-27) In the foregoing analysis, it was

demonstrated that, contrary to OSHA's contention regarding the inapplicability of ventilation to ETS, ASHRAE Standard 62-1989 for Ventilation was specifically designed for, and has been proven successful in, the dilution and removal of ETS constituents. The Standard currently provides ventilation design/operation criteria for building codes in the U.S.

A precursor standard to ASHRAE 62-1989, ASHRAE Standard 62-1973, recommended a ventilation rate of 15 cfm outside air/person, comparable to the minimum recommended outdoor air rate specified in ASHRAE 62-1989. Standard 62-1973 was approved by the American National Standards Institute (ANSI) and incorporated into building codes. It was in effect through the 1970s. ASHRAE updated Standard 62-1973 in 1981 and provided for two ventilation rates based on smoking and nonsmoking (Standard 62-1981). That Standard did not receive approval from ANSI and was not incorporated into the major building codes in the U.S. ASHRAE 62-1973 thus remained in effect throughout the 1980s as the design and operational criteria document for ventilation in building codes. Many HVAC systems designed and installed over the past 20 years have complied with the specifications in ASHRAE 62-1973 and ASHRAE 62-1989.

The ASHRAE Standards (62-1973 and 62-1989) designate the required outdoor air portion of total supply air, where total

supply air equals outside air and return air. The minimum outdoor rates specified in the Standards actually constitute only a fraction of the supply air needed to provide proper heating and cooling; outside air supply requirements constitute a small percentage of the air needed for total supply air. If outside air specifications differ from Standard to Standard, overall HVAC capacities, as determined by minimum design criteria for supply air, would satisfy the demand for greater outdoor air flow rates. Indeed, recent research on 160 office buildings by Sundell, et al. indicated that buildings of different age categories did not differ operationally with regard to outdoor air flow rates.[25] The assumption that an increase in outside air ventilation rates would require the redesign and retrofit of an existing ventilation system is not supported.

Differences in minimum outside air requirements between the two versions of the ASHRAE Standard are not likely to be significant and may, in fact, be identical for certain indoor areas. **Thus, compliance with the Proposed Rule's requirements for minimum outdoor air ventilation rates for IAQ, insofar as they are based on the 1973 and 1989 versions of the ASHRAE Ventilation Standard, would be sufficient to satisfy the ventilation requirements for the effective dilution and removal of ETS constituents under ad lib smoking situations.** There are no feasibility constraints based on compliance with current building

code specifications for ventilation, as specified in OSHA's general ventilation recommendation for IAQ.

As OSHA's Proposed Rule on ETS tacitly admits, an enclosed, separately exhausted and negatively pressurized smoking room is neither technologically nor economically feasible as a real option available to all of the nation's employers. (59 FR 16016, 16013) As demonstrated above, negative pressurization is feasible using existing exhaust and supply locations in a building. A requirement for a separate exhaust from a smoking area directly to the outside is burdensome, superfluous and dictated only by OSHA's "zero exposure" doctrine for ETS. Scientific studies and IAQ reports indicate that smoking and nonsmoking areas may share a common air handling system with recirculation of air such that constituents from ETS from smoking areas are not significantly redistributed to nonsmoking areas. This condition can be achieved if the building conforms to the specifications for ventilation rates and filtration recommended by the current ASHRAE Standard for ventilation -- indeed, by OSHA's own Proposed Rule on IAQ.

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APPENDIX A

THE BUILDING SYSTEMS APPROACH

The Building Systems Approach To Acceptable Indoor Air Quality

A Review and Analysis of OSHA RFI Docket H-122: "Occupational Exposure to Indoor Air Pollutants"

This paper presents an analysis of what has come to be known as the "building systems approach" for the prevention and mitigation of poor indoor air quality (IAQ). Supporting data for the analysis provided in this text were obtained through a review of the public comment submissions to the OSHA RFI Docket on Indoor Air Pollution (H-122).

The review of the RFI docket on indoor air included all public comments received by OSHA through April of 1992. Consideration was given to all "substantive" comments, i.e., comments which included either references to the published literature on IAQ or to observations about indoor air quality. Opinions which were not supported by data, experience or even anecdotal information were not considered "substantive," and were excluded from consideration.

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IAQ Building Systems Approach

Overview: The Building Systems Approach is Based Upon the Proper Design, Operation, and Maintenance of a Building's HVAC System.

The building systems approach to achieving and maintaining acceptable indoor air quality emphasizes attention to the proper design, operation and maintenance of a building's heating, ventilation and air-conditioning (HVAC) system. The approach is advocated by a substantial number of respondents to the OSHA RFI, representing such diverse groups as governmental agencies (Wisconsin Dept. of Industry and Labor, 3-10; Kansas Dept. of Human Resources, 3-1094; University of California, 3-1192); labor unions (the AFL-CIO, 3-1185; Food and Allied Services Trades, 3-434; Am. Fed. of State, County and Municipal Employees, 3-446; Int. Fed. of Professional and Technical Engineers, 3-770); professional organizations and trade associations (the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE), 3-440; the Business Council on Indoor Air (BCIA), 3-933; the National Energy Management Institute (NEMI), 3-1183; the Tobacco Institute, 3-1086; the Sheet Metal and Air Conditioning Contractors National Association (SMACNA), 3-856; the National Environmental Development Association's (TIEQ), 3-1054); IAQ monitoring and mitigation experts (T.D. Sterling and Associates Ltd. (TDSA), 3-1073; Healthy Buildings International (HBI), 3-1053; Gershon Meckler Associated, 3-879; Alan Hedge, 3-955; James Woods, 3-745; Eagle Environmental Health, 3-500; Terracon International, 3-501), and product manufacturers (Philip Morris, 3-1074; R.J. Reynolds, 3-1087; Johnson Controls, 3-1102 and Dow Chemical, 3-502).

In its simplest terms, the central focus of the building systems approach is upon appropriate ventilation, which, in turn, includes provisions for adequate outdoor supply air and its distribution to occupied spaces. The intended effect of this focus is to decrease exposure to substances in indoor air and to prevent and/or alleviate complaints about indoor air quality. (BCIA, 3-933; Dow, 3-502) This is a performance-orientated, "engineering systems" approach which requires the inter-disciplinary expertise of architects, mechanical engineers, HVAC contractors and maintenance engineers.

The approach recognizes the existence of potential "significant risk" and/or "material impairment" from worker exposures to poor indoor air quality, as documented by literally thousands of building investigations completed in the past decade. It acknowledges that individual causes of complaints related to indoor air quality in "problem buildings" are rarely identifiable, and that suspected causes of poor indoor air quality are likely to be multi-factorial, involving any number of substances at low-exposure levels, as well as comfort-related factors such as air movement, temperature, lighting and humidity. (TDSA, 3-1073; HBI, 3-1053; NEDA-TIEQ, 3-1054; Hedge, 3-955)

The approach offers a comprehensive solution to poor indoor air quality. Adequate supply air intake and its distribution serve to dilute and/or remove a wide range of potential substances in the indoor air, including volatile organic compounds (VOC's) such as benzene and formaldehyde, carbon monoxide (CO), carbon dioxide (CO_2), constituents of environmental tobacco smoke (PTS), radon, biologicals, etc. (ASHRAE, 3-440; NEMI, 3-1183; PM, 3-1074) This solution remains constant as the work activities of building tenants and the "mix" of substances in indoor air change over time. The effectiveness of the building systems approach is adequately supported by data in the published literature, and will be discussed at length below. (See: "D. Ventilation: The Mitigation Procedure for Poor IAQ.")

The buildings systems approach is also a *feasible* solution to indoor air quality problems and to the maintenance of acceptable indoor air. The approach relies upon already existent technology and standards for the design, operation and maintenance of HVAC systems. It is likewise a *cost-effective* solution, because any costs associated with compliance with the approach are likely to be small and offset by a reduction in complaints and a potential increase in worker productivity. (TDSA, 3-1073; PM, 3-1074; BCIA, 3-933; Woods, 3-745; Ventresca, 3-941) The approach directs resources to controlling the widest range of potential substances in indoor air.

The Scientific Basis For the Building Systems Approach

The basic tenets of the building systems approach are:

(A) Indoor air quality problems are most often associated with inadequate HVAC system performance, including incorrect system design (improper sizing, modification and/or installation), inadequate outside air intake, poor filtration and/or inadequate preventive maintenance of the system; (Eagle EH, 3-500; NEMI, 3-1183; HBI, 3-1053);

(B) Complaints regarding unacceptable indoor air quality (usually associated with symptoms of irritation, annoyance and discomfort) cannot, as a rule, be directly traced to specific airborne substances or to levels of exposure; (TDSA, 3-1073; PM, 3-1074; Hedge, 3-955; HBI, 3-1053);

(C) A number of environmental factors unrelated to IAQ may also contribute to complaints about indoor air quality. These include temperature, humidity, lighting, noise, and physical "stressors" such as workload, job satisfaction and general ergonomic conditions; (Hedge, 3-955; TDSA, 3-1073; NEDA-TIEQ, 3-1054; NEMI, 3-1183; AFL-CIO, 3-1185);

(D) Remediation and mitigation of poor indoor air quality, and the attainment and maintenance of acceptable indoor air quality, depend upon the correct design, operation and maintenance of a buildings' HVAC system. (ASHRAE, 3-440; BCIA, 3-933; NEMI, 3-1183; HBI, 3-1053).

The basic premises for the building systems approach are directly supported by data from over 2,000 individual building investigations conducted by governmental and private agencies in the U.S. and Canada. These building investigation reports are included in eight large independent databases on "sick-building syndrome," and can be found in the published literature on IAQ. (PM, 3-1074)

The individual sick-building databases exhibit remarkable similarities in their reported results. IAQ complaints in roughly one half (or more) of the building investigations have been related to inadequate ventilation. An additional (and often substantial) percentage of the complaints reported in the investigations have been associated with poor HVAC maintenance, including poor filtration and contaminated systems. Specific airborne substances have been identifiable as "causal factors" for complaints in fewer than 20 percent of all building investigations.

Inadequate Ventilation is Associated With Approximately Half of All Complaints in Sick-Building Cases

Building investigation reports in the published literature fall under the heading of "sick-building syndrome" investigations, wherein complaints of irritation and discomfort (e.g., headaches, eye, nose, and throat irritation, dizziness, shortness of breath, mental fatigue, drowsiness, and lethargy) are reported, and IAQ specialists/building investigators are asked to determine potential causal agents and possible remedial actions. (Eagle EH, 3-500; HBI, 3-1053; TDSA, 3-1073; AFL-CIO, 3-1185)

One of the most notable compilations of building investigations was undertaken by NIOSH and referenced in the OSHA RFI (*Fed. Reg.* 56(183), September 20, 1991). Inadequate ventilation was identified as a primary problem in fifty-two percent of the 484 building investigations in the NIOSH database. Inside contamination was identifiable in only fifteen percent of all buildings; microbiological contaminants were identified in five percent.[1,2] (PM, 3-1074)

A database of 1,362 building investigations compiled by Health and Welfare Canada also identified inadequate ventilation as the primary problem in 52 percent of the buildings investigated. Specific indoor air contaminants were identified in only 12 percent of the investigations.[3,4] (TDSA, 3-1073; PM, 3-1074)

Public Works Canada, another Canadian federal agency, investigated 30 buildings for IAQ complaints between

1987 and 1990. Ventilation-related problems were reported in one half of the buildings.[5] (TDSA, 3-1073)

TDSA Ltd. have compiled data on 408 building investigations conducted in the U.S. and Canada. The results have been analyzed and computerized in what is called the "Building Performance Database." Ventilation-related inadequacies were directly associated with IAQ complaints in 49 percent of the buildings catalogued in the Building Performance Database.[6] (TDSA, 3-1073)

A private, U.S.-based IAQ monitoring firm conducted 412 sick-building investigations from 1981 through 1988. Ventilation problems were associated with complaints in 62 percent of the buildings investigated; bacterial and fungal contamination was reported in nearly a third of all buildings investigated. (HBI, 3-1053)

A 1989 report by Dr. James Woods assessed 30 cases of "problem buildings" investigated by the Honeywell Corporation since 1986. Woods' research indicates that 75 percent of the buildings investigated had inadequate outdoor supply air intake. Similarly, 75 percent of the buildings exhibited inadequate air distribution to occupied spaces, and 65 percent of the buildings suffered from inadequate HVAC maintenance.[7] (Woods, 3-745; PM, 3-1074)

Kim (1990) summarized 105 investigations of problem buildings undertaken by Clayton Environmental Consultants, a IAQ monitoring firm in the U.S.[8] (Morey, 3-505) She wrote:

> In a survey of 105 buildings, Clayton found that 53 percent had [HVAC] maintenance problems, 49 percent had operational problems (such as improper handling of control equipment), and 33 percent had design problems. Mechanical engineers evaluated the HVAC systems in 70 of the buildings, in which they found that 75 percent had maintenance problems, 70 percent had operational problems, and 47 percent had design problems. Of the 105 buildings, 95 were sampled for contaminants. Of these, 28 were found to have problem levels of microbial contaminants, 26 had volatile organic compounds and 13 combustion products. (PM 3-1074)

Freund et al. from the New Jersey Department of Health evaluated 221 complaint buildings in that state and reported that 43 percent were associated with inadequate ventilation.[9] (HBI, 3-1053)

The Oregon Department of Resources submitted results of 36 state office building investigations to the OSHA RFI (3-1157). The respondents observed:

> Thirty-six state office buildings were examined for IAQ problems. Significant problems were found in 16 buildings. Many of the problems were associated with inadequate ventilation and high levels of carbon dioxide . . . studies in Oregon have shown that when outside make-up air falls below 15 cfm, complaints increase.

In a submission to the OSHA docket by Local 12/ Occupational Illness Support Group of the U.S. Department of Labor, the authors report that:

> Inadequate ventilation is the primary cause of most of the indoor air quality problems . . . . This is a result of the Department's inability to maintain adequate amounts of outside air . . . . (3-1017)

Published data from a number of sick-building investigation databases reveal that deficiencies in ventilation have been directly related to complaints about IAQ in one half or more of all reported cases. HVAC-related problems have been associated with as many as 75 percent of the complaints in several of the sick-building databases discussed above.

Significant Risk Associated With Poor IAQ: Poor IAQ in the Workplace is a Widespread Problem; Data from Building Investigations Indicate a Significant Risk of Impairment for Non-Industrial Workers Associated With Poor IAQ; Costs to Employers and Workers

In 1982, the World Health Organization described a complex of eight symptom groups which could be used to characterized sick-building syndrome (SBS). Those symptoms included: irritation of the eyes, nose and throat; dry mucous membranes in skin; erythema; mental fatigue and headache; cough; wheezing; nausea

and dizziness; and hypersensitivity reactions. (TDSA, 3-1073) In addition, SBS has also been characterized by complaints of discomfort which do not precisely fit any of the WHO's eight categories, e.g., "stuffiness," chills, unpleasant odors, etc. (Hedge, 3-955)

Building Related Illness (BRI), as indicated earlier, describes infirmities of known cause which are often traceable to sources of microbial contamination in the building or to the transmission of infectious respiratory disease. Although specific, documented cases of BRI are rare in comparison to complaints about poor indoor air quality, there is perhaps little doubt that BRI poses a potential significant risk for workers in the non-industrial workplace. Support for this statement can be derived from the sick-building syndrome databases. For example, Public Works Canada identified microbial problems in 16 percent of the 30 buildings they investigated.[5] Robertson of HBI reported allergenic fungi in 34 percent of buildings investigated and allergic or pathogenic bacteria in 9 percent of the buildings. (HBI, 3-1053)

The submission to the OSHA RFI docket by TDSA Ltd. includes an estimate of between 26,000 and 116,000 annual cases of sporadic Legionnaires disease in the United States. (TDSA, 3-1073) Other kinds of communicable and non-communicable building-related infections exist, ranging from hypersensitivity pneumonitis to the flu. However, data to support a precise numerical projection of total cases are apparently not available.

Symptoms of irritation and discomfort reported in thousands of sick-building syndrome investigations strongly suggest the existence of an additional potential significant risk of "impairment" for non-industrial workers. Sick-buildings, of course, constitute only a subset of the total non-industrial workplace building stock in the United States. Nevertheless, the World Health Organization and others have estimated that SBS may affect as much as 30 percent of all buildings in the U.S., which in turn may affect 20-30 percent of the non-industrial workforce. (Woods, 3-745)

The AFL-CIO has estimated in its response to the OSHA RFI that indoor air pollution may effect "30-70 million building occupants and between 800,000 and 1,200,000 commercial buildings in the United States . . ." (AFL-CIO, 3-1185) A substantial number of other respondents to the OSHA RFI provide estimates of risks attributable to poor IAQ, which are summarized in Appendix I of this document.

Poor indoor air quality also imposes additional costs upon employers and workers alike. Such costs include those related to health and the costs of lowered productivity, stemming from greater absenteeism, loss of concentration, etc. Although such purported costs can only be estimated from the available data, respondents to the OSHA RFI contend that they are indeed substantial — perhaps amounting to billions of dollars annually. (BCIA, 3-933) (For a complete analysis of costs attributed to poor IAQ by respondents to the OSHA RFI, see Appendix II.)

Data on BRI and SBS thus verify the potential significant risk posed by poor IAQ on the non-industrial workforce in the United States. Potentially burdensome costs to the American employer and worker alike are similarly associated with incidents of BRI and SBS.

IAQ Complaints Generally Cannot be Associated With Specific Airborne Substances

The OSHA RFI on Indoor Pollutants sought information and data on a number of substances potentially associated with complaints about IAQ. These substances include: volatile organic compounds (VOC's), biologicals, passive tobacco smoke (PTS), carbon monoxide (CO), carbon dioxide (CO_2), and radon. (For a review of the RFI docket submissions which discuss these substances, see Appendix III.) A review of the docket indicates that while these substances are often mentioned as potential "causes" of poor IAQ, few data exist which suggest that *specific* substance exposures have been associated with *specific* complaints. Moreover, the databases on sick-building syndrome support the contention that specific airborne substances rarely are identifiable as sources of IAQ problems or complaints. A number of comments in the RFI docket substantiate the above claims. For example:

The Atlantic Richfield Company (ARCO) reports that, in their experience, "when monitoring has been

conducted, hazardous contaminants have either not been detected, or they are present in concentrations far below those known to present health hazards." (ARCO, 3-448)

The submission from Organization Resources Counselors states that:

> Companies report monitoring for formaldehyde, total and respirable particulates, total organics. . . . In almost all cases where monitoring was done for specific contaminants, results were either below the level of detection, or were below OSHA Permissible Exposure Limits. (3-1084)

Similarly, CanTox, Inc. reports that:

> More than half (63 percent) of the compounds detected in indoor air could not be attributed to a definite source. The largest group with known sources were found to have multiple sources and their presence could not be exclusively attributed to one specific source. This clearly has significant implications with respect to attempting to use source control to maintain indoor air quality. (3-1180)

The American Federation of Government Employees (AFGE) of the AFL-CIO reports that "unfortunately, despite the substantial evidence linking poor indoor air quality to AFGE members' adverse health effects, AFGE is unable to obtain the data needed to make the causal connection between specific contaminants and those adverse health effects." (3-529)

United Technologies reports that, "based on our experience in occupational settings and knowledge of the professional literature, there are only very weak data that directly relate specific chemicals to IAQ." (3-651)

TDS and Associates Ltd. report, after citing four studies, that:

> The correlation between symptoms presented in IAQ complaints and causative agents is weak because exposure to many different types of contaminants in indoor air, originating from both indoor and outdoor sources, has been shown to result in similar health and comfort complaints. The presence of pollutants in indoor air, combined with thermal comfort parameters of temperature and humidity, and other building characteristics, such as ventilation, lighting, noise and occupant density and activities, make it difficult to isolate the causative agent and IAQ related health and comfort complaints. (3-1073)

The American Industrial Hygiene Association (AIHA) notes that, based on their experience:

> In most IAQ complaints, symptoms are non-specific and could be caused by a variety of factors. Correlations can be found where there is a consistent spacial and temporal relationship and the complaint can be resolved by changing the building condition. Biological contamination may sometimes be distinguished by a pattern of allergy symptoms. (3-735)

The Philip Morris submission to the RFI docket cites a study by Canadian researchers that reported results of a building investigation in which temperature, humidity, dust, nicotine, formaldehyde, volatile organic compounds, and CO_2 were measured.[10] Occupants of the building complained of poor indoor air quality, and a questionnaire evaluation revealed a substantial number of complaints about unsatisfactory thermal conditions, dry air, drowsiness and eye irritation. However, all measured parameters, including temperature and humidity, were within accepted comfort exposure guidelines. The authors were unable to correlate any single measured environmental parameter with complaints or symptoms. They concluded that their investigation "showed that complaints reported by occupants were associated with perceived rather than measured levels of indoor environmental parameters." (PM, 3-1074)

The Building Performance Database compiled by T.D. Sterling and Associates Ltd. provides data on airborne substance monitoring from well over 200 sick-building investigations. Average recorded levels of carbon dioxide, carbon monoxide, respirable suspended particles, formaldehyde, airborne fungi and bacteria, nicotine, and temperature and humidity, are all within parameters of "acceptable" exposure. (See Appendix IVa.) Nevertheless, the buildings from which the monitoring results arose were deemed "sick." The authors conclude: "In general, IAQ investigations of white collar workplaces have found indoor concentra-

tions of measured substances far below occupational exposure levels. . . ." (TDSA, 3-1073)

PTS Workplace Exposures: The SBS Databases Reveal that PTS May Be Directly Associated With Complaints in Only Two to Four Percent of All Investigations; Data from Workplace Exposure Studies on PTS Constituents Indicate That Typical Nonsmoker Exposures to PTS are Minimal and Often Below Analytical Limits of Detection

Because it is visible and easily identified by its aroma, passive tobacco smoke (PTS) is often initially identified as the cause indoor air quality problems. However, data submitted to the OSHA RFI on actual workplace exposure levels to PTS constituents, together with conclusions derived from several major databases on sick-building syndrome, indicate that tobacco smoke is rarely the underlying *cause* of complaints about poor indoor air quality. For example, in HBI's database of 412 sick-buildings, ETS was reported to be a significant contributor to complaints in only 3 percent of all buildings investigated. (HBI, 3-1053) In the sick-building database compiled by TDSA Ltd., smoking was implicated as a major contributor to complaints in only 12 of 408 (less than 3 percent) of the buildings surveyed.[6] (TDSA, 3-1073) NIOSH investigated more than 200 "sick-buildings" through 1984 and reported that tobacco smoke was a source of claimed discomfort in only 2 percent of the buildings investigated.[1] (PM, 3-1074) In a summary of 94 building studies by government investigators from Health and Welfare Canada, complaints were attributable to indoor constituents such as photocopy machine emissions and PTS in only 5 percent of the buildings investigated.[3] (PM, 3-1074) In all of the databases, inadequate ventilation was determined as the underlying source of complaints in over half of all investigations.

It is nevertheless understandable, given the easy recognition of PTS, that persons experiencing sick-building symptoms initially tend to blame PTS. Indeed, researchers have indicated that the mere visibility or presence of tobacco smoke may provoke claims that PTS is the cause of reported symptoms and complaints.[11,12] This is likely to be especially true when an individual is already feeling discomfort in a SBS setting. (PM, 3-1074) However, as one commentator has pointed out: "Removing the smoker entirely, then, would not effect health and comfort problems in 95 to 98 percent of sick-buildings."[13] (TDSA, 3-1073)

Moreover, published data submitted to the RFI docket on the contribution of PTS constituents to overall ambient air levels in typical workplaces suggest that such contributions are minimal and often indistinguishable from contributions from other sources.[14] (ORNL, 3-499; TDSA, 3-1073; Reasor, 3-1071; Holcomb, 3-1065) Of course, data from monitoring studies on PTS-related constituents in the workplace are varied and depend on a number of factors, such as the number of smokers present, activity levels, ventilation, total volume, etc. (See Appendix IVb.) Nevertheless, it is possible to observe trends in the data. For example, after a review of 37 supporting studies from the published literature, Philip Morris reports that:[15-51]

> . . . [T]here is little difference in ambient levels of carbon monoxide in smoking and nonsmoking areas of workplaces and public places and in homes with and without smokers. Other studies indicate that PTS contributes approximately 30 percent of the total particles in the air of a typical public place. Nicotine is often used as marker for PTS exposures because it is unique to tobacco smoke. Typical measurements of nicotine range from an exposure equivalent of 1/100 to less than 1/1,000 of one filter cigarette per hour. This means that a nonsmoker would have to spend from 100 to 1,000 hours or more in an office, restaurant, or public in order to be exposed to the nicotine equivalent of a single cigarette. (3-1074)

Philip Morris also observes that "studies which have examined PTS constituent levels of nitrosamines, nitrogen oxides and volatile organic compounds (such as benzene) report minimal contributions to overall ambient air levels in homes, the workplace, and public places."[26,28,44-51] (3-1074)

In the most comprehensive review and analysis of the literature on ETS exposure published to date, researchers from Oak Ridge National Laboratories (ORNL) observe that "it has been difficult to discern the effect

of CO from ETS in any situation except extreme conditions of experimentally designed exposure."[14] (ORNL, 3-499)

The ORNL monograph on ETS also reports that "indoor concentrations of NO, NO_2, or NO_x can be somewhat elevated when compared to outdoor background, but show very little dependance upon the presence of ETS." The authors also note: "It has been difficult for many studies to demonstrate consistently elevated levels of formaldehyde due to ETS."

The ORNL monograph also reviews and discusses studies on the measurement of tobacco smoke particles (RSP's) in the workplace which have employed UV/PM (ultra-violet particulate matter) methods of analysis. Studies indicate the following RSP contributions from ETS; e.g., (1) 131 offices—27 ug/m3; (2) 22 offices—32 ug/m3; (3) 194 offices—28 ug/m3; and (4) 31 offices—44 ug/m3.

The ORNL monograph concludes that:

> Studies of offices, restaurants, train compartments, and public buildings suggest that ETS contributes to the indoor air burden of VOC's but that other sources predominate. Major sources include building materials, furnishings, cleaning products, office machines, gasoline, and combustion sources associated with cooling, heating, and transportation. (ORNL, 3-499)

Other respondents to the OSHA RFI reach similar conclusions. Dr. Mark Reasor, after a review of over 25 published studies, concludes that "ETS is unlikely to contribute significantly to daily workplace exposures to volatile organic compounds, carbon monoxide, oxides of nitrogen, polycyclic aromatic hydrocarbons, and nitrosamines," and that "it may influence exposure to nicotine and to respirable suspended particulates, but under adequate ventilation conditions, this exposure is likely to be low." (Reasor, 3-1071)

The submission by Theodor D. Sterling and Associates, Ltd. reports results of PTS constituent monitoring in workplaces undertaken by TDSA.[16,22,42] The data indicate the existence of extremely low levels of nicotine — sometimes at or below the detection limits of the analytic method — and concentrations of RSPs ranging from 10 to 50 ug/m^3. RSP levels reported in nonsmoking workareas were low (17-20 ug/m^3), and there were no significant differences reported between areas that did or did not receive ventilation air recirculated from smoking lounges. (See Appendix IVb.) (TDSA, 3-1073)

Professor Alan Hedge of Columbia University reported the results of his study on 4,479 office workers from 27 air-conditioned offices. Hedge examined the potential impact of smoking and smoking policies on reports of sick-building syndrome. With the exception of slightly elevated levels of nicotine in non-smoking offices adjoined to smoking areas, monitoring results for various PTS constituents revealed no significant differences in workareas where smoking was permitted compared to workareas where smoking was not permitted. (Hedge, 3-955)

HBI submitted data from their study of 585 offices in which various PTS-related constituents were monitored.[41] (HBI, 3-1053) Aside from increased airborne levels of nicotine in smoking offices, the levels of ETS-related constituents reported in typical smoking and nonsmoking areas were statistically indistinguishable. For example, the average level of carbon monoxide in 254 nonsmoking offices was 3.1 ppm; the average for 331 smoking offices was 3.4 ppm. Similarly, the average level of particulates (RSP ug/m^3) recorded for 331 offices in which smoking was permitted was 46 ug/m^3. The average for 254 nonsmoking offices was 20 ug/m^3. The authors note that these "real-life respirable particulate particle levels averaged about four times lower than the Repace and Lowrey model predicted, at 46 ug/m^3." They also observe that the average levels of particles in both smoking and nonsmoking workareas were substantially less than the level designated as "Limited or No Concern" (100 ug/m^3) by the World Health Organization's "Consensus of Concern for Tobacco Smoke Particles."

Thus, a review of the scientific data on exposure levels to ETS in the workplace, together with analyses of those studies provided by respondents to the OSHA RFI, indicate that typical workplace levels of ETS constituents cannot be associated with a significant exposure risk to nonsmoking workers.

PTS: Reports of Chronic Health Effects in the Workplace are not Supported by the Available Epidemiologic Data

The scientific literature reporting measurements of PTS constituents in indoor air provides little support for claims about possible chronic health effects among nonsmokers in the workplace. Moreover, the available data from published epidemiologic studies on the workplace do not provide sufficient support for the claim that PTS exposure in the workplace is associated with chronic disease in nonsmokers. (See Appendix III; 3. PTS.)

A primary deficiency in the epidemiologic studies on PTS is the lack of reliable exposure data. Published epidemiologic studies which report associations between spousal smoking and chronic disease (e.g., lung cancer, heart disease, etc.) in nonsmokers are not based on actual exposure assessments for ETS. Instead, these studies rely on subjective responses to questionnaires to assess "exposure" rather than on any quantifiable measurement. Questionnaire inquiries regarding "spousal smoking" or "living with a smoker" are used as surrogate determinants for ETS exposure in these studies. Recent studies indicate, however, that such subjective assessments are an extremely unreliable and inaccurate measure of exposure.[52] Furthermore, questionnaire responses about exposure often vary widely when compared with actual measurements of ETS constituents in the workplace.[53]

Lung Cancer

The argument that PTS exposure increases the risk of lung cancer in nonsmokers is based on an interpretation of the data from epidemiologic studies of nonsmoking women married to smokers (spousal smoking). Of the 33 published epidemiologic studies on the issue of spousal smoking and lung cancer, none actually *measured* exposure to PTS. Moreover, twenty-seven of the 33 studies report results which are not statistically significant overall — that is, their conclusions were consistent with the null hypothesis of no association between reported exposure to PTS and lung cancer in nonsmokers. Most important, however, is that fourteen of the published spousal studies also assessed reported *workplace* exposures to ETS via questionnaire (although no actual measurements were conducted).[54] Twelve of the 14 workplace studies report associations between ETS and nonsmoker lung cancer which do *not* achieve statistical significance. Only two studies report marginally statistically significant increased risks for persons who reported exposure to ETS in the workplace.

Heart Disease

There are no studies in the published literature which have examined actual ETS exposures in the workplace and heart disease in nonsmokers. Only twelve epidemiologic studies on spousal smoking in the home and heart disease in nonsmokers are available. These studies, based on marriage to a smoker, are not directly relevant to the workplace issue. In addition, the spousal smoking studies on heart disease contain no data on actual exposures to ETS. Instead, exposure estimates are derived from questionnaire responses.

Nonetheless, five of the 12 published epidemiologic studies on spousal smoking and heart disease did attempt to address workplace exposures to ETS.[55] None of these studies reported a statistically significant increased risk of heart disease among nonsmokers claiming exposure to ETS in the workplace. Thus, the existing literature does not provide support for the claim that ETS exposure in the workplace is related to an increase in risk of heart disease among nonsmokers.

Respiratory Disease Other Than Cancer/Asthma

Few studies have examined the issue of exposure to ETS in the workplace and respiratory disease (other than cancer) in nonsmoking adults.[56] The studies that have been conducted on this issue have reported contradictory results. Researchers who have reviewed these studies have cited problems with the methodology utilized and have pointed out that confounding factors (e.g., other exposures) were not considered.[57]

Data on the possible association between ETS exposure and increased risk of an asthmatic reaction are similarly inconsistent. There are ten major studies on the possible association between exposure to ETS and acute respiratory symptoms in adult asthmatics.[58] The studies are inconsistent in their reported results which range from reported decreases in pulmonary function to no acute respiratory effects among asthmatics

exposed to ETS. The studies that do report an association between adult asthma and exposure to ETS suffer from several methodological flaws that include: (1) confounding factors that are not adequately controlled for in many studies; (2) inadequate sample sizes; (3) psychological factors that have not been ruled out; and, (4) reliance on unrealistic exposure conditions in enclosed smog chambers.

Thus, the available published epidemiologic data on reported PTS exposures in the workplace and various chronic disease endpoints in nonsmokers do not support the claim that PTS poses a significant risk for chronic disease among workers. This is not to deny that some individuals may nevertheless report annoyance or irritation by the sight or smell of PTS. The building systems approach to achieving and maintaining adequate indoor air quality is designed to address those kinds of complaints.

Other Environmental Variables Unrelated to IAQ Play a Role in Worker Complaints About Health and Comfort

Advocates of the building systems approach recognize the existence of a number of non-IAQ environmental variables which influence worker perceptions of health and comfort. Hedge et al. have proposed a model which identifies five kinds of variables involved in the determination of sick-building syndrome. (Hedge, 3-955)

These include:

1. Individual factors (age and gender);
2. Organizational factors (type of job);
3. Architectural and engineering factors (type of ventilation, office design, controls/access);
4. Psychosocial factors (stress, satisfaction);
5. Non-IAQ environmental factors (thermal conditions, lighting, noise).

A number of studies in the published literature indicate that lighting, temperature, humidity, job satisfaction, job stress and ergonomics are factors which will influence the kinds of worker perceptions about poor/adequate IAQ. (TDSA, 3-1073) For example, in a SBS study in Denmark, Skov and colleagues examined sick-building syndrome reports among 4,369 office workers. Their research indicated that indoor climate perception was strongly related to the prevalence of SBS symptoms. Lifestyle factors were only weakly associated with the reporting of symptoms.[59] (PM, 3-1074)

A 1991 report by Hawkins and Wang ranked a number of variables related to self-reported symptoms of sick building syndrome.[60] Those variables included: "humidity," "satisfaction with work," "active smoking," "gender," "exposure to PTS," "office light," and "doing professional work." They concluded:

> Building Sickness Score was associated with many factors. Sick building syndrome symptoms are influenced by multiple variables of which the environmental factor of humidity and he psychological factors of work, sex, and occupation are important. (PM, 3-1074)

Based on one of his own studies, Hedge reports that although ventilation has an important effect on indoor air quality, reports from workers in 46 office buildings in the U.K. indicated that complaints were even more strongly influenced by a number of personal and occupational factors such as gender, job stress, job satisfaction and computer use. (3-955)

The American Industrial Hygiene Association (AIHA) reports that "psychosocial and physical stresses are certainly potential causes of some IAQ complaints and should always be considered in any investigation." This opinion is based on the experience of AIHA members. (3-735)

Citing two studies, Eagle Environmental Health reports that "thermal discomfort, unpleasant odors, lack of air movement, insufficient lighting, and excessive noise are also indicated in IAQ investigations. Job-related stress many also manifest itself in IAQ complaints." (3-500)

The National Energy Management Institute (NEMI) reports:

> The NEMI experience has revealed that there are a variety of factors which can interact to cause a worker to display indoor environmental health-related problems. These factors may actually be the primary cause or may exacerbate an IAQ problem condition. Temperature, temperature change, humidity, air velocity, light levels, noise as well as psycho-social factors

should *always* be considered in presenting and investigating IAQ problems. (3-1183)

The U.S. EPA also recognizes that factors related to SBS are multi-factorial, involving combined environmental and psycho-social stressors. (3-1075; Attachment H) Referencing the World Health Organization (1986), the U.S. EPA notes:

> Buildings at highest risk [of SBS] appear to be new or recently remodeled buildings with tight envelopes, especially those with large ventilation systems that depend on limited fresh air sources. Improper ventilation, thermal conditions, and occupant lack of control over climatic and working conditions are other factors that may increase the likelihood of a building being linked to sick-building syndrome. (3-1075; Attachment E)

A NIOSH psychologist, Dr. Michael Colligan, has offered an explanation for the role of such factors in perceptions about IAQ.[61] (PM, 3-1074) He writes:

> It appears then, that the individual is sensitive to fluctuations in the functioning of the autonomic nervous system. When perceived changes in his subjective state are understandable, e.g., 'I have an allergy,' 'I've been under a lot of pressure to met a deadline,' 'I'm worried about my teenager,' an individual can initiate various coping strategies to deal with the causes. When the origins of the experienced distress are vague or unclear, however, an individual starts searching around for salient cues. If the environment provides a plausible cause in the form of a pungent odor or dense, stuffy air, then an individual can conclude, rightly or wrongly, that the poor quality of the environment is responsible for his physical and psychological discomfort. Notice that this process can occur independently of any specific toxic effects the environment might have on the individual and irrespective of the 'real' cause of the autonomic arousal. All that is required is that individual experience autonomic arousal in response to a subtle or unidentified stressor or combination of stressors. cues provided by the environment in the form of noxious odors, visually detectable particulates or dust, or humid, stuffy air, may suggest to an individual that his discomfort is a toxic response to an airborne pollutant. That environment then becomes a source of threat to the individual, which in turn may generate more autonomic arousal and anxiety.

Dr. Colligan's observations provide an understanding of complaints related to PTS in the workplace. Because it is readily identifiable, PTS is often *initially* blamed for IAQ problems (yet after investigation, reported exposures to PTS are directly associated with complaints in only 2-4 percent of sick buildings). If individuals are "stressed" by their work environment (influenced by such diverse factors as temperature, humidity, air movement, ergonomics, workload, personal problems, etc.), the mere visibility of PTS may provide a cue for a complaint. Recent research by Winneke and colleagues indicates that an individual who is predisposed to annoyance from PTS will be more likely, under actual exposure conditions, to respond with annoyance symptoms.[12] (PM, 3-1074) Researchers from the Illinois Institute of Technology also addressed the issue of nonsmoker perception of annoyance and irritation from PTS exposures. The study, involving over 250 subjects, reported that visual contact with a smoker affects the magnitude of adverse response to PTS among selected nonsmokers. The authors suggest that their conclusion "provides an inexpensive strategy of reducing complaints associated with PTS: eliminate visual contact between smokers and nonsmokers." (IIT, 3-31)

Precise quantitation of psychological variables and perceptions of comfort within the context of complaints about IAQ is extremely difficult. Nevertheless, the influence of those environmental variables upon IAQ complaints should be considered during SBS investigations. The comfort parameters of humidity, temperature and air distribution are related to HVAC performance, and the building systems approach is capable of addressing those factors.

Ventilation: The Mitigation Procedure for Poor IAQ

Ambient concentrations of substances measured in the non-industrial workplace and reported in the published literature rarely approximate occupational PEL or TLV levels. Moreover, specific substances are rarely identifiable as causes of complaints about poor IAQ. Nevertheless, such complaints are presumed to be "real" (i.e., they may present physiological manifestations) and they may be said to constitute some degree of "material impairment" of the worker.

What remedial and/or preventive measures would reduce non-industrial worker low-level ambient exposures and complaints about IAQ? According to the building systems approach, the appropriate mitigation procedure begins with increased attention to ventilation, and specifically, to adequate supply air, distribution and air filtration. The RFI docket is replete with such recommendations. For example, the New York State Building and Construction Trades Council reports that, based on their experience, "the average office setting exposes workers to contaminants from machines, carpets, paints, glues, and fungi. These contaminants mix with the air the workers breathe on a daily basis and affect a persons well-being. Proper ventilation has been shown to provide a proven antidote to these problems." (3-732)

IAQ investigators from AFSCME Local 12 from the University of Iowa Employees Union report that "we have found that insufficient fresh air flow is most often the cause of a number of symptoms, including coughing, skin and eye irritations, headaches and upper respiratory infections . . . indoor air quality problems can be treated with little effort and expense by improving or upgrading inadequate ventilation systems to increase fresh air flow in the workplace." (3-809)

Investigators from CanTox Inc. agree: "[C]ontrol of the air exchange rate (i.e., ventilation) of a building is probably the most important and practical mediation practice for management of indoor air quality." (3-1180)

The State of New Jersey Health Department's investigation of 221 complaint buildings revealed that over 43 percent of all cases involved inadequate ventilation. In cases where abatement recommendations were made, the recommended strategy consisted of increased maintenance, repair, adjustment or redesign of the HVAC system. Eighty-four percent of the cases where such abatement steps were implemented reported an elimination of complaints." (HBI, 3-1053)

Indoor air quality in schools has recently become an issue of considerable concern. In 1989, Helsing and coworkers reported the results of an IAQ investigation in a school. They reported that "there was an insufficient fresh air supply to some classrooms and a large percentage of students exhibited classic symptoms of sick-building syndrome, i.e., headache, eye burning, fatigue."[62] Similarly, investigations by Hanssen (1987)[63] and Beller (1989)[64] reported that low air exchange rates in combination with installation of new building materials was the main cause of complaints in the schools that they investigated. Helsing et al. concluded: "Correcting the ventilation problems resulted in reduction of symptoms to a level approximately equal to that of students in other schools in the county." (PM, 3-1074)

In a 1991 publication, Thompson et al., described various retrofit projects for HVAC systems in 26 schools across the U.S.[65] The retrofits followed the design specifications set forth in ASHRAE Standard 62-1989 for ventilation. The retrofits were, according to the authors, "very effective" at reducing radon and CO_2 levels in the schools. The authors also reported that "many of the occupants" believed that IAQ had improved. (PM, 1074)

The State of Wisconsin Safety and Building Division reports that, in their experience:

> Almost without fail, . . . complaints are resolved by ventilating the offices or classrooms per state code. This is a provision of outside air (up to 20 cubic feet per minute) per person. . . . These ventilation requirements are supported by the American Society of Heating, Air-Conditioning, and Refrigerating Engineers (ASHRAE). (3-10)

In 1989, Collett and Sterling examined the effect of ventilation retrofits on perceived health and comfort complaints by building occupants. For the buildings in which major retrofits were undertaken, perceptions of indoor air quality improved in seven of nine categories surveyed. (TDSA, 3-1073)

Downing and Bayer recently reported results from more than 35 building IAQ investigations.[66] They reported that the most "common source of IAQ problems has been the lack of proper operation and maintenance (O&M) of buildings." They further observed that "in more than 80 percent of the investigations to date, changes in building O&M significantly improve the perception of IAQ by the occupants." The researchers recommend, as part of their O&M procedure check list for indoor air quality, "to raise outdoor air ventilation to ASHRAE recommended minimums." (PM, 3-1074)

In 1991, investigators of the U.S. EPA's headquarters in Washington, D.C., reported that they were "unable to establish consistent relationships between major environmental parameters and self-reported health symptoms among the sampled employees." However, based on the number and frequency of complaints among workers in those buildings, the research group recommends that "an attempt to maintain indoor environment in accordance with the ASHRAE guidelines should be made."[67] (PM, 3-1074)

Similarly, Canadian health officials who investigated and compiled the Canadian sick-building database (Health and Welfare Canada) observed that recommendations for improvements in ventilation and thermal comfort had been made in 60 percent of the buildings 1,400 investigated, while control of specific pollutants was recommended in only 20 percent of all cases.[68] (PM, 3-1074)

In 1984, a committee on sick-building syndrome from the WHO's Europe Working Group on Indoor Air Research concluded that an increase in outdoor supply rates, together with improvements in air distribution, had satisfactory results in remediating sick-building syndrome. (Lebowitz, 3-188)

The Ohio Civil Service Employees Association, after reviewing three indoor air quality incidents in Ohio, endorsed the ventilation approach "as a vital component in solving and preventing persistent health and safety problems related to indoor air in the workplace." (3-398)

Steven B. Hayward of the State of California Department of Health Services recommends adoption of a minimum ventilation standard similar to that currently in effect as a Cal/OSHA regulation. The Cal/OSHA standard requires that a minimum supply of outdoor air specified in the State Building Standard Code be provided, and that the HVAC system be operated continuously, inspected regularly, and properly maintained. (3-17A)

A former NIOSH building investigator now associated with a public health department in New York State remarked that "indoor air quality problems are widespread in buildings throughout the state. In almost all cases, the cause of complaints in a given building can be readily identified. In most cases, the problem is the ventilation system for the building. Most often these complaints are due to exposure to multiple substances which are not being adequately exhausted from the building due to inadequate ventilation." (NY Pub. Emp. Fed., 3-444)

Respondents to the RFI also observe that adequate ventilation is the preferred method for controlling exposures to radon, VOC's, PTS, CO, $C)_2$,and bioaerosols. (Eagle EH., 3-500; Burge, 3-61; HBI, 3-1053) Indeed, the U.S. Department of Labor's Occupational Illness Support Group states that PTS and radon . . . "have seldom been the source of employee complaints of poor indoor air quality. With properly designed and properly operated ventilation systems, exposure to radon and passive smoke can be minimized." (3-1017)

Feasibility (Technological and Economic) and Docket Endorsement of Ventilation Rates Prescribed for Various Workplace Settings in ASHRAE Standard 62-1989

Ventilation is the cornerstone of the building systems approach to acceptable indoor air quality. Air changes are required in structures to replace air depleted by human respiration, to remove excess humidity and to dilute and remove airborne substances which are released by indoor sources. Various studies and testimonials submitted in response to the OSHA RFI provide verification for the use of a ventilation approach in the control of indoor air quality.

According to submissions to the OSHA RFI docket, the most widely endorsed guidelines on ventilation have been promulgated by the American Society of

Heating, Refrigeration, and Air-Conditioning Engineers, Inc. (ASHRAE), a private, standards-writing organization. ASHRAE is a professional organization consisting of architects, mechanical and HVAC engineers, HVAC systems manufacturers, contractors and IAQ mitigation experts. One of the principal functions of ASHRAE is the development of consensus standards relating to heating, ventilation and air-conditioning. ASHRAE develops and promulgates standards which are adopted by building code organizations in the United States. (ASHRAE, 3-440)

In 1989, ASHRAE published a standard entitled "Ventilation for Acceptable Indoor Air Quality."[69] The standard is known as ASHRAE Standard 62-1989 and prescribes the rate at which fresh or outside air must be delivered to occupied spaces, and the various means to condition that air. In 1990, Standard 62-1989 was approved by the American National Standards Institute (ANSI). The Standard has been adopted in twelve states and by two major building code organizations in the United States. It is currently under consideration for adoption by the remaining national building code organizations. (PM, 3-1074)

ASHRAE Standard 62-1989 establishes ventilation rates and procedures for various indoor settings in order to "control dioxide and other contaminants with an adequate margin of safety, and to account for variations among people, varied activity levels, and a moderate amount of smoking."[69]

The ventilation rates recommended by ASHRAE 62-1989 are comparable to rates prescribed for indoor areas in Finland, Sweden, Japan, and Germany. The Standard also accords with the September 1988 draft recommendations by the International Committee on Indoor Air Quality for the providence of Ontario, Canada. That committee recommended a ventilation rate higher than 15 cfm/pp for all "offices, schools and similar non-industrial buildings." (PM, 3-1074)

ASHRAE Standard 62-1989 also recommends limits of exposure to various constituents found in indoor air. The upper limits for exposure are 1/10th of the threshold limit values (TLV's) prescribed by the ACGIH.[69]

The precursor ventilation standard to ASHRAE Standard 62-1989, ASHRAE 62-1981, recommended two levels of ventilation, one for areas in which smoking was permitted, and another substantially lower rate for areas where smoking was prohibited. The prescribed ventilation rate in ASHRAE 62-1981 for offices in which smoking was permitted (20 cubic feet outside air per minute per person; 20 cfm/person) was 4 times greater than the rate recommended for nonsmoking areas (5 cfm/person).

IAQ problems were reported by those who followed the minimum (nonsmoking) ventilation rates specified in ASHRAE 62-1981. Research indicates that the ventilation rate for nonsmoking areas (5 cfm/person) is insufficient to efficiently dilute carbon dioxide and body odor and that occupants under such conditions may complain of "stuffy" air. Other research indicates that a ventilation rate of at least 15 cfm/person, the minimum rate recommended by ASHRAE Standard 62-1989, and three times the rate recommended for nonsmoking areas by ASHRAE 62-1981, would be necessary and sufficient to disperse normally occurring ambient substances (e.g. CO_2, body odors, etc.), as well as tobacco smoke.[70-72] (ASHRAE, 3-440)

In recent testimony before a Congressional Subcommittee, Mr. John Janssen, the Chairman of the ASHRAE Standards Project Committee for 62-1989, stated:

> Prior to the oil embargo of 1973, buildings tended to be over-ventilated and indoor air quality problems were not widely recognized. Ventilation recommendations for office spaces, for example, ranged all the way from 5 to 25 cubic feet per minute per occupant. In 1973, ASHRAE published the first edition of Standard 62, which allowed a minimum of 5 cfm of outdoor air per person for some applications. Most state and city building codes still reference ASHRAE Standard 62-1973, "Standard for Natural and Mechanical Ventilation."
>
> In 1981, the ASHRAE Ventilation Standard was revised to incorporate new technology and new awareness of such issues as tobacco smoke, which was not mentioned in the 1973 version. Research results (some ASHRAE sponsored) showed that the minimum ventilating rate of 5 cfm per occupant permitted under 62-73 would not adequately control